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Biomechanics and Electromyography of a Common Idiopathic Low Back Disorder

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Study Design. *In vivo* feline preparation groups loaded into lumbar flexion at different magnitudes and an unloaded control group.

Objective. To demonstrate that a static, constant load flexion of the lumbar spine results in a complex neuromuscular disorder.

Summary of Background Data. Epidemiology suggests that static lumbar flexion is a cause of low back disorders. There is little direct experimental evidence describing the physiologic and biomechanical processes that elicit the disorder. Recent evidence shows that static flexion of the spine under constant displacement results in muscular spasms and a prolonged recovery period. The response of the spine to flexion under constant load of various magnitudes (as opposed to constant displacement) is not known. It was hypothesized that static lumbar flexion under constant load may elicit creep in spinal ligaments, discs, *etc.*, causing microdamage and development of a neuromuscular disorder.

Methods. The lumbar spine of the feline was subjected to 20 minutes of constant load static flexion at physiologic load intensities from light to heavy while creep of lumbar viscoelastic tissues and EMG from the multifidus muscles of L3–L4 to L5–L6 were recorded. Recordings were continued over a 7-hour rest period after the static flexion was terminated.

Results. Spasms and decreasing reflexive EMG were evident during the loading period, and a transient surge of EMG activity occurred at the beginning of the rest period. A second surge of EMG activity was seen 3–4 hours later. The four components of the neuromuscular disorder were present regardless of the load magnitude. A model was developed to quantify the disorder.

Conclusion. A four-component neuromuscular disorder was elicited by a 20-minute constant load static flexion even when very light loads were applied. The disorder was elicited by creep of the viscoelastic tissues, which resulted in spasms and muscular hyperexcitability lasting for >24 hours. Although the disorder was transient, the physiologic and biomechanical principles associated with its development could also explain cumulative trauma disorders. [Key words: low back disorder, ligament, EMG, muscle, pain] **Spine 2003;28:1235–1248**

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Musculoskeletal disorders (MSDs) have become a major health concern resulting from the industrial revolution and technological advances.[1,2] In 1994, the U.S. Department of Labor, Bureau of Labor Statistics, determined that the number of injuries or illnesses resulting from static and repetitive motion and overexertion was 705,800.[2] The National Institute for Occupational Safety and Health recently estimated the cost of MSDs at $13 to $20 billion annually.[3] In 1997, approximately 472,000 back, spine, and spinal cord cases were recorded in different industries, including service (28%), manufacturing (21%), and retail (16%).[4]

Risk factors for MSDs comprise four major categories: genetic, morphologic, biomechanical, and psychological.[1] Although genetic and morphologic factors play a prominent role in the development of MSDs, only biomechanical and psychological risk factors allow interventional preventive control strategies. Among the various biomechanical risk factors, exposure to repetitive, static, and vibratory activities are known to result in MSDs.[1] This study focuses on the impact of static lumbar loading on the development of MSDs in the lumbar spine.

Static load applied to ligaments results in creep (*e.g.*, stretch of viscoelastic tissue over time which is not fully restored immediately after load removal).[5–10] Theoretically, ligaments that remain stretched beyond their resting length may result in increased laxity of intervertebral joints and risk of excessive relative displacement, instability, and possible injury.[4,11]

In the spine, ligaments have a secondary role in maintaining intervertebral stability,[12–14] and the musculature is the major structure responsible for this function.[13,15,16] However, it was shown that ligaments and discs are endowed with afferents,[17–19] and a reflex exists from these afferents to the lumbar multifidus and longissimus muscles[20–23] providing synergistic ligamentomuscular action to keep the lumbar joints stable.

Passive cyclic[24,25] and static[26,27] displacement-controlled loading of the lumbar spine into flexion results in reflexive activity of the multifidus, which decays exponentially with time as tension–relaxation of the vis-

coelastic tissues sets in. In static flexion, large spasms are also superimposed on the EMG discharge from the multifidus, especially when the static displacement is kept for more than 2–3 minutes. In essence, the decrease in reflexive muscular activity together with the loss of tension in the ligaments render the spine unprotected to excessive intervertebral motion, which may result in injury. The spasms suggest that tissue damage was present.[18]

Distinct differences exist between constant displacement and constant load as the input stimuli to the spine.[28] Previous studies only used constant passive displacement as the input stimulus,[24–27] so the response to constant load is still unknown. Furthermore, when a load-controlled stimulus is applied, the outcome would be expected to be dependent on the load magnitude.

The objectives of this study were as follows: 1) to determine the response of the lumbar spine to various constant static loads, within the physiologic range; and 2) to develop a model for lumbar displacement (creep) and reflexive muscular activity behavior during static loading and the following recovery. Specifically, it was of interest to assess the development of creep within the spine's viscoelastic structures (*e.g.*, ligaments, discs, and capsules) and the behavior of reflexive muscular activity associated with the various static magnitudes of constant loads and during 7 hours of recovery (rest) after the static load was removed. The outcome of this study may provide significant insights into the physiologic and biomechanical behavior of the lumbar spine subjected to the physiologic range of static loads, the mechanisms that may elicit MSDs, and possible preventive interventions.

■ Methods

Preparation. Twenty-seven adult cats (4.00 ± 0.4 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 level to the sacral level and allowed to retract laterally to expose the dorsolumbar fascia. After dissection, the preparation was placed in a rigid stainless steel frame that allowed the isolation of various lumbar levels by external fixation (discussed in the next section). A gauze pad soaked with saline was applied over the incision during 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 of the L3–L4, L4–L5, and L5–L6, 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 of 110 dB common mode rejection ratio, a gain capability of up to 200,000, and a band pass filter of 6–500 Hz. EMG responses from each channel were monitored on oscilloscopes and stored in a computer at a sampling rate of 1000 Hz.

An S-shaped stainless steel hook, made of a 1.5-mm rod, was inserted around the middle part of the L4–L5 supraspinous ligament and connected to the vertical actuator of a Bionix 858 Material Testing System (MTS, Inc., Minneapolis, MN). The load was applied by the MTS actuator with a computer-controlled loading system operated in load control mode. The vertical displacement of the actuator and the load cell output incorporated in it were also sampled into the computer along with the EMG data.

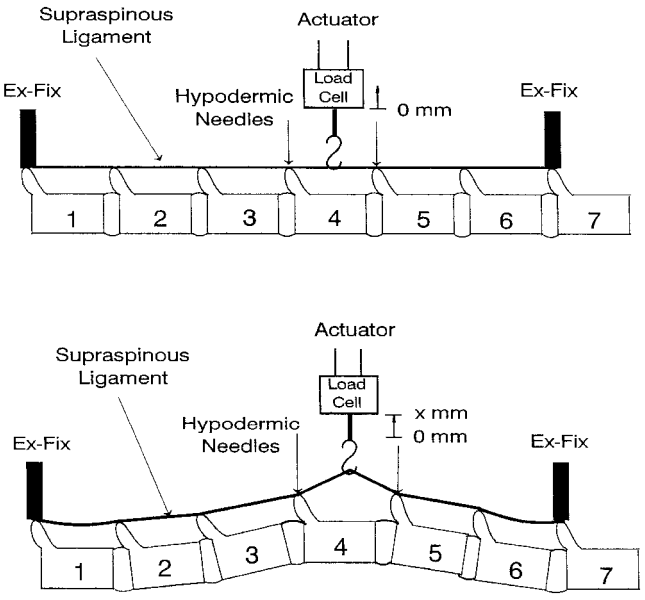

Figure 1. Schematic of the experimental setup showing the lumbar spine in the initial condition (top) and during the sustained static flexion (bottom). SSL = supraspinous ligament.

Two external fixators were used to isolate the lumbar spine: a first fixator to the L1 posterior spinal process and a second fixator to the L7 process. The external fixation was intended to limit the elicited flexion to the lumbar spine and to prevent interaction of thoracic and sacral–pelvic structures. The intention of the external fixation was not, however, to prevent any motion. A schematic of the experimental setup is shown in Figure 1.

Protocol. The stainless steel hook applied to the L4–L5 supraspinous ligament was pulled up by the MTS actuator from a resting position with a preload of 1 N applied just before a 20-minute static load period, immediately after the 20-minute static load period was terminated, and immediately after a 7-hour rest period. Vertical displacement (in mm) at L4–L5 supraspinous ligament was measured from MTS actuator sensor on each occasion when the tension was 1 N. Two short hypodermic needles were inserted into the spinous processes of L4 and L5 to mark the length of the supraspinous ligament. The length of the supraspinous ligament between these two needles was measured by using a digital electronic caliper immediately before and immediately after the 20-minute load application and at the end of the 7-hour rest period, while the static tension was reset to 1 N. The vertical displacement values at 1 N load and L4–L5 supraspinous ligament length were used to estimate the creep in the L4–L5 supraspinous ligament. Electromyograms from the three multifidus muscles, load, and displacement were recorded continuously during the 20-minute loading period.

During the 7 hours of rest, 8-second tests were performed to assess vertical displacement and EMG recovery. Tests were applied after 10 minutes of rest, 30 minutes of rest, 60 minutes,

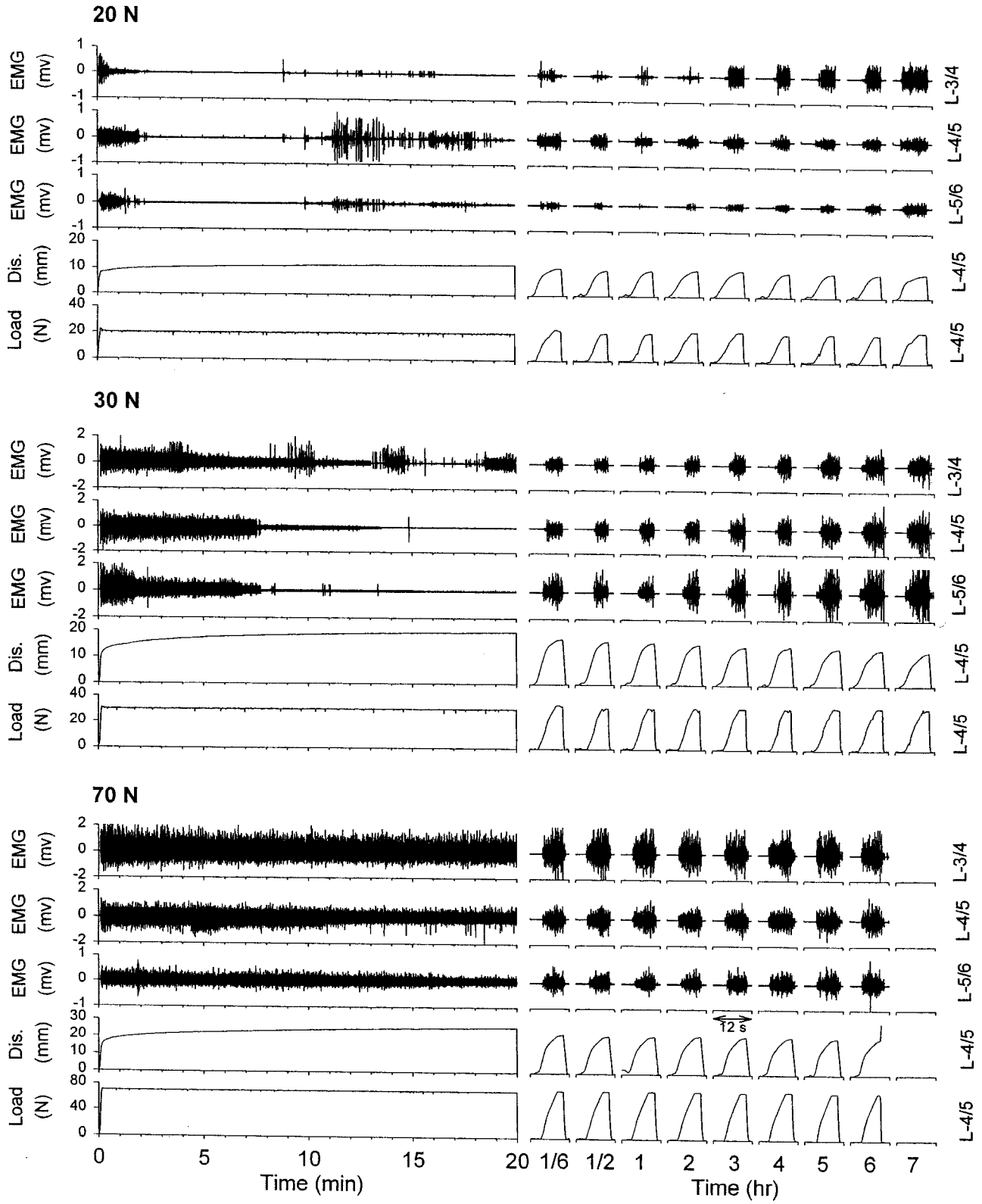

Figure 2. Typical recording of EMG, lumbar spine displacement, and constant load during the sustained flexion as well as the short tests during the 7-hour recovery period for 20 N, 30 N, and 70 N loads.

and each hour thereafter. Each 8-second test consisted of a 6-second linear increase in load to the maximal used in that test followed by 2 seconds of constant load. The spine remained unloaded between the specified tests (see typical tests in Figure 2). The rapid unloading did not affect the EMG activity, as it silenced immediately. A slow rate of increase in load was used to avoid inflicting damage to the ligaments as is known to occur with the exposure to a sudden or fast stretch.[29–31] Similarly,

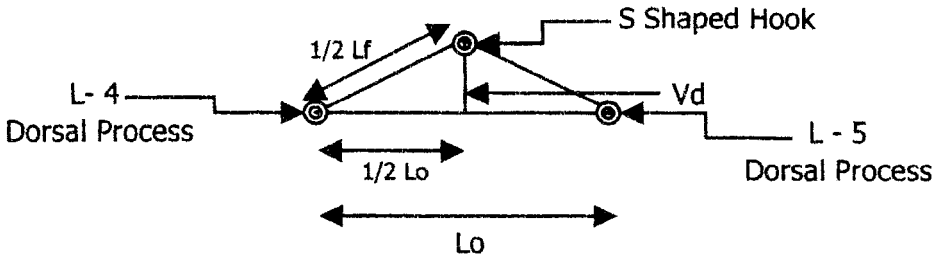

Figure 3. Schematic of the L4–L5 supraspinous ligament subjected to 1 N constant load for purposes of measurement of the creep and residual creep.

the load was increased linearly to its maximal value in the initial 6 seconds of the 20-minute loading period. Each 8-second test was recorded in a 12-second window triggered by the computer at the appropriate time.

The same protocol was used for each of the four static loads of 20 N (n = 5), 30 N (n = 6), 50 N (n = 4), and 70 N (n = 7). The load values covered the complete range from the reflex excitation threshold load of the ligament (20 N) to just below the maximal load sustained by the ligament (70 N) as determined in pilot studies. The values for creep (at 20 minutes) and residual creep (at the end of 7-hour recovery) were calculated separately for each of the loads applied. Five preparations were used as controls. In the control set of animals the dissection, application of the external fixation, EMG electrodes, and S-shaped hook around the supraspinous ligament were performed as usual. The animals, however, were not subjected to loads and were left undisturbed for the same period (20 minutes, plus 7 hours). This was done to assure that the applied load was indeed the source of any neuromuscular disorder recorded.

Analysis. Windows (1.5 seconds) of electromyogram, static load applied to the spine, and vertical displacement at the L4–L5 supraspinous ligament were sampled immediately at the beginning of the loading period and every 20 seconds thereafter for the 20-minute static loading, as well as for the short tests in the recovery period. Each electromyogram sample was integrated over the 1.5-second window and normalized with respect to the value obtained from the first window. The normalized integrated EMG (NIEMG) of all the preparations subjected to the same load at the respective window were pooled, and the mean and standard deviation were calculated and plotted on a NIEMG *versus* time plot for each of the preparations. Similarly, displacements of the respective windows of all preparations subjected to the same load were pooled, and the mean and standard deviation were calculated and plotted as displacement *versus* time plot.

The length of the supraspinous ligament was measured at 1 N preload before and immediately after the 20-minute load was applied and immediately after the 7-hour recovery period. These values and the associated vertical displacement of the supraspinous ligament were used to calculate the creep and residual creep, respectively, in the ligament by using equations (1) and (2), derived from Figure 3.

$$Lf = 2\sqrt{\left(\frac{1}{2}Lo^2\right) + Vd^2} \quad (1)$$

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

Where Lo is the distance between the two hypodermic needles inserted into L4 and L5 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.

Model Development. The pooled NIEMG data 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 and/or elongation. The model structure for NIEMG and actuator displacement in the loading period takes the form shown in equations (3) and (4), respectively.[26,32]

For the NIEMG:

$$NIEMG(t) = Ae^{-t/T1} + NIEMG_0 \quad (3)$$

Where
A = exponential component initial amplitude;
$T1$ = exponential decay time constant;
$NIEMG_0$ = steady state NIEMG amplitude;
t = time.

The displacement followed an exponential model as follows:

$$DISP(t) = D_0 + D_L(1 - e^{-t/T2}) \quad (4)$$

Where
$DISP(t)$ = actuator vertical displacement as a function of time;
D_0 = elastic component of displacement;
D_L = viscoelastic component amplitude;
$T2$ = time constant;
t = time.

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

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

$$DISP(t) = D_c + D_R e^{-t/T3} \quad (5)$$

Where D_c = displacement at the end of the 20-minute loading;
D_R = recovery of the creep over 7-hour rest;
$T3$ = recovery time constant;
For the NIEMG, the model format was as follows:

$$NIEMG(t) = E(1 - e^{-t/T4}) + tBe^{-t/T5} + C(tt - Td)e^{-(t - Td)/T6} + NIEMG_0 \quad (6)$$

Where $E(1-e^{t/T1})$ = the steady state recovery component;
$tBe^{-t/T2}$ = a transient hyperexcitability component;

$C(t-Td)e^{-(t-Td)/T6}$ = a delayed transient hyperexcitability ("morning after");

$NIEMG_0$ = the residual response at the end of the 20-minute constant load.

In this model, the constraint of $E + NIEMG_0 = 1$ is used to ensure that full recovery results in a normal (unity) response.

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

■ Results

Figure 2 shows typical EMG responses from each of the three lumbar levels as well as load and displacement for the 20-minute loading period and 7 hours of recovery from preparations subjected to 20 N, 30 N, and 70 N loads.

In general, the recorded data demonstrated that the EMG activity decreased gradually during the 20-minute loading period for all the preparations exposed to the different loads. Spasms, visualized as spontaneous electromyogram discharge with various amplitudes and frequencies, occurred in different lumbar levels regardless of the load magnitude. Also, the timing of the spasms varied widely. In some of the preparations, the spasms occurred for a brief period of time and diminished after 2 to 12 minutes. In the others, the spasms appeared to be continuous for the whole 20-minute static loading period for different lumbar levels. The lumbar levels in which electromyographic spasms were recorded, the timing, duration, and the amplitude showed a random behavior.

During the recovery period, the EMG recorded from the first 8-second test was relatively large compared with that of the following three or four tests, which demonstrated decrease. At the end of the recovery period, the typical EMG was larger than that recorded at the beginning of the 20-minute loading period. In some preparations it was 2 to 3 times larger in peak-to-peak amplitude.

The typical displacement patterns demonstrated an exponential-like increase (which represents the viscoelastic tissue creep associated with constant load) during the 20-minute loading period. During the recovery, the peak displacement gradually decreased, indicating that viscoelastic tissues were recovering toward their resting dimensions.

Figure 4 provides the mean ± SD of the NIEMG recorded from the three lumbar levels and the mean ± SD displacement recorded for the pooled data from the tests at 20 N, 30 N, 50 N, and 70 N during the 20-minute loading. Figure 5 provides the same for the 7 hours of recovery. For the set subjected to 70 N load, in two preparations the L4–L5 supraspinous ligaments ruptured. One ruptured during the 20-minute test and the second during the last test (7th hour of recovery). The data from the preparation which ruptured during the 20-minute test were excluded from the analysis, whereas the data from the second preparation were included except of the last data point, resulting in n = 6 for the 70 N test.

The control preparations (n = 5) did not exhibit any changes in baseline EMG throughout the testing period. No spasms or any other unusual discharge were recorded, confirming that the applied load was the source of the responses (described above) in the test preparations.

L4–L5 Supraspinous Ligament Creep

The L4–L5 supraspinous ligament creep at the end of the 20-minute loading test and at the end of 7 hours of recovery, as calculated from equations (1) and (2), is shown in Table 1. For the preparations subjected to a 20 N load, the supraspinous ligament was 6.01% longer at the end of the 20-minute test and then returned to 4.38% over its resting length after the 7-hour recovery, a reduction of 27.1%. Similarly, the preparations subjected to 20-minute loads of 30 N, 50 N, and 70 N exhibited gradually increasing mean creep values of 12.37%, 13.1%, and 22.25%, respectively. Surprisingly, after the 7-hour rest, the residual creep in the ligaments had recovered by 43.6%, 56.9%, and 66.4%, for the 30 N, 50 N, and 70 N groups, respectively. In essence, larger recovery was seen for preparations exposed to larger loads. Nevertheless, the residual creep after rest was, in general, larger for higher loads. A residual creep of 4.38% was present in the group subjected to a 20 N load, whereas 7.48% residual creep was present in the group subjected to a 70 N load.

The creep in the L4–L5 supraspinous ligament did not fully recover after 7 hours in any of the preparations used in this study, indicating that it is not a sufficient interval to allow full recovery of the creep developed in 20 minutes of static flexion. About half of the creep developed during the 20-minute static flexion was still present.

Lumbar Spine Displacement

The vertical displacement of the lumbar spine at L4–L5 during the application of the constant loads in the 20-minute test and in the 8-second tests during recovery is an indirect measure of the overall shear creep developed in the viscoelastic tissues of the spine. These include various ligaments (supraspinous, intraspinous, anterior and posterior longitudinal) in all lumbar levels, as well as capsules and discs of all lumbar levels. The creep developed in the L4–L5 supraspinous ligament is only one of the many components included in the shear creep measurement represented by the vertical displacement. Figures 4 and 5 show the mean displacement throughout the experiment for each of the four loads applied, and Table 2 provides the initial mean vertical displacement and mean displacement at the end of the 20-minute test and after 7 hours of recovery for each of the four load intensities used in this study.

In general, the displacement demonstrated an exponential rise throughout the 20-minute exposure to static load and an exponential decrease throughout the recov-

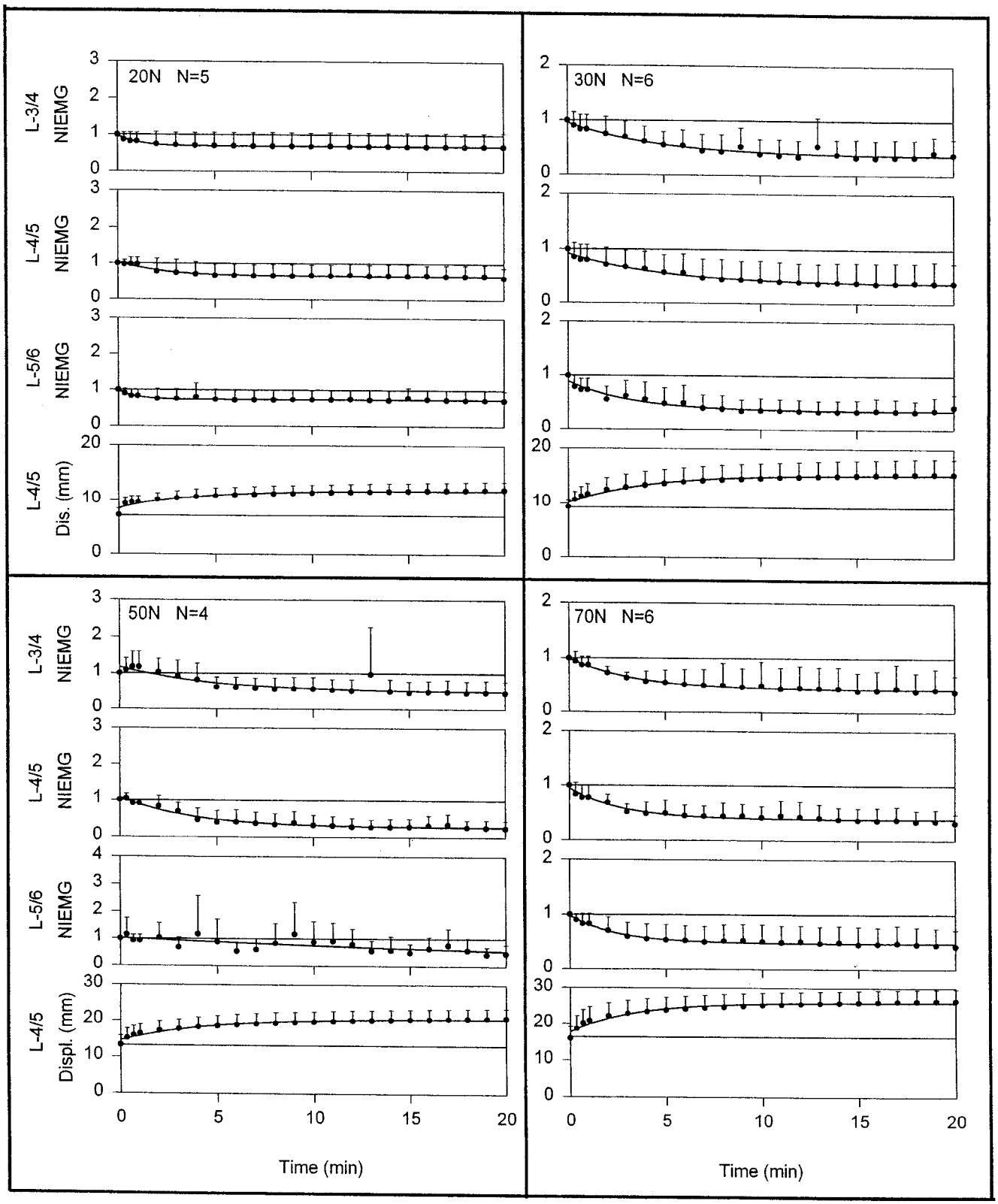

Figure 4. The normalized mean (± SD) of the NIEMG and mean (± SD) displacement of the pooled data for each of the four loads applied to the lumbar spine during the 20-minute static load period.

ery. For the set exposed to a load of 20 N, the mean initial displacement was 7.18 ± 0.12 mm and at the end of the 20 minutes it was 12.1 ± 1.43 mm, a 68.5% increase. At the end of the 7-hour rest period, the displacement recovered to 9.76 ± 1.37 mm, indicating that it was still 35.9% over its initial value.

For the preparations exposed to the 30 N load, the mean initial displacement was 9.24 ± 1.39 mm, and at

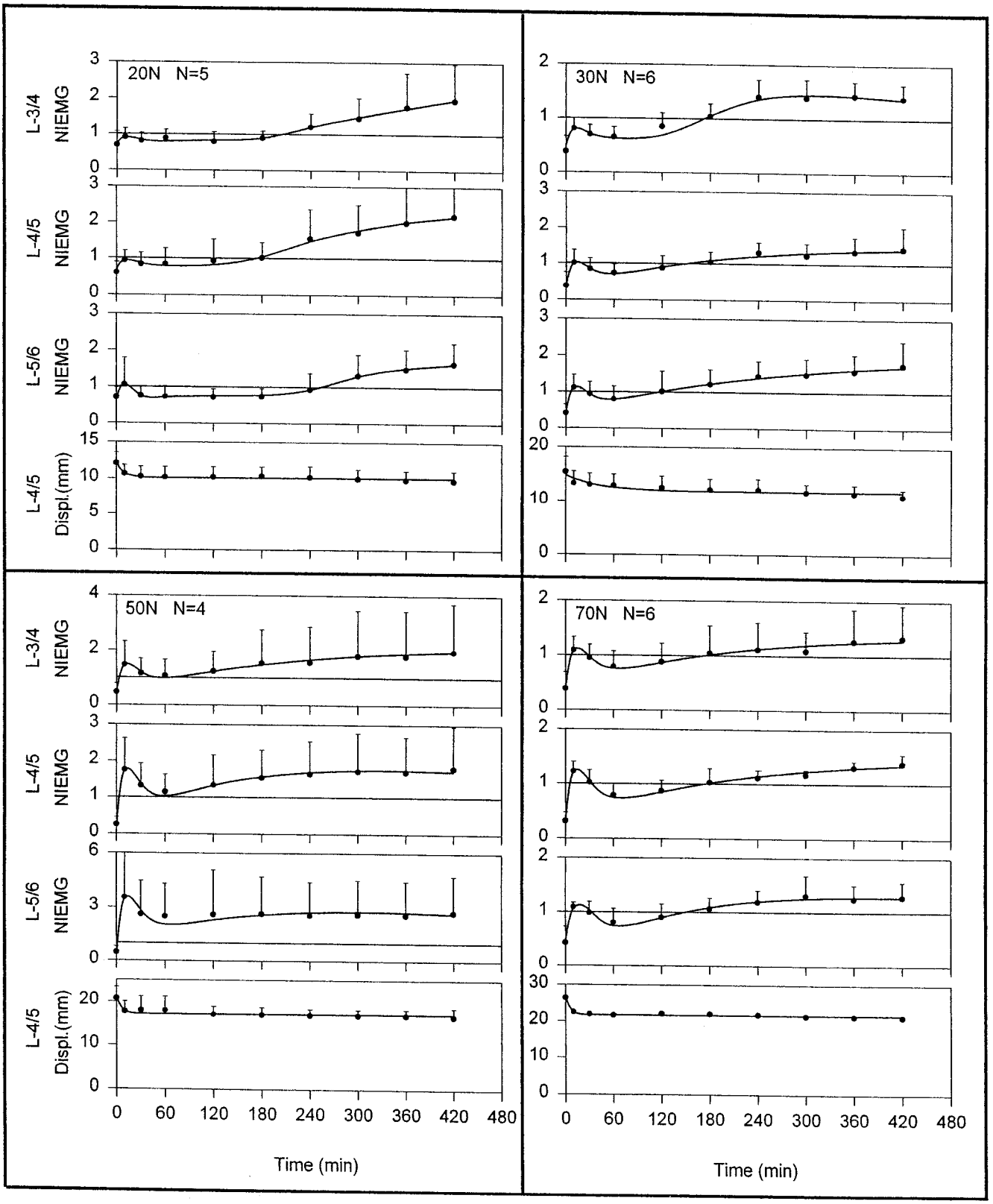

Figure 5. The normalized mean (± SD) of the NIEMG and mean (± SD) displacement of the pooled data for each of the four loads applied to the lumbar spine during the 7-hour recovery period.

the end of the 20 minutes it was 15.5 ± 2.66 mm, a 67.7% increase. After 7 hours of rest, the residual displacement was 11.1 ± 1.25 mm, or 20.2% over its initial value.

For the set exposed to the 50 N load, the mean initial displacement was 13.2 ± 2.61 mm, and at the end of the 20-minute flexion, it was 20.8 ± 2.66 mm, a 57.4% increase. Seven hours of rest allowed a recovery to 16.6

Table 1. L4/L5 Supraspinous Ligament Creep

Load N	Mean Creep at End of 20 Mins	Mean Creep at End of 7 Hrs	% Recovery
20 N	6.01 ± 1.52%	4.38 ± 2%	27.1
30 N	12.37 ± 5.4%	6.98 ± 4.9%	43.6
50 N	13.1 ± 5.07%	5.65 ± 2%	56.9
70 N	22.25 ± 3.6%	7.48 ± 6.7%	66.4

± 2.02 mm, which is still 25.8% over its initial displacement.

For the set loaded to 70 N, the mean initial vertical displacement was 16.0 ± 3.15 mm, and at the end of 20 minutes, it increased by 65.5% to 26.5 ± 3.22 mm. After 7 hours of rest, it recovered to 21.2 ± 3.18 mm, which is still 32.4% over its initial length.

In general, larger loads resulted in larger initial vertical displacement, yet the percent increase in vertical displacement at the end of 20-minute static flexion was not affected by the load magnitude, ranging from 57.4% to 68.5%. Similarly, the residual displacement at the end of the recovery period varied from 35.9% over the initial displacement at the 20 N load, to 20.2%, 25.8%, and 32.4% for loads of 30 N, 50 N, and 70 N, respectively. The load magnitude did not seem to have an impact on the residual displacement as well. Complete recovery of the vertical displacement to its initial value was never observed in any of the preparations.

EMG Response

The mean NIEMG values of the multifidus muscles of the five preparations subjected to the 20 N load (Figure 4, top left) demonstrated an exponential decrease for the three lumbar levels over the 20-minute loading period, decreasing to 69%, 61%, and 71% of the initial EMG for L3–L4, L4–L5, and L5–L6, respectively. Spasms were present throughout the 20-minute loading period, appearing in each preparation at random and variable magnitudes and frequencies. There was transient hyperexcitability in the first 30 minutes of recovery in all lumbar levels. At the end of the first 10-minute recovery period, the mean NIEMG values of the multifidus muscles peaked, demonstrating increases to 91%, 94%, and 105% of their initial value for L3–L4, L4–L5, and L5–L6, respectively. The NIEMG decreased after the first 10 minutes of rest to the end of the first hour. During the remaining 6 hours of rest, the mean NIEMG values gradually increased to 195%, 220%, and 225% in L3–L4, L4–L5, and L5–L6, respectively.

The mean NIEMG values for the multifidus muscles of the six preparations loaded to 30 N (Figure 4, top right) demonstrate an exponential decrease for the three lumbar levels during the 20-minute loading period, decreasing to 38%, 38%, and 42% of the initial EMG for L3–L4, L4–L5, and L5–L6, respectively. Spasms were observed in each of the preparations during the 20 minutes. In the recovery period, there was a transitory hyperexcitability during the first hour of recovery. The mean NIEMG values of the multifidus muscles peaked at 81%, 100%, and 112% of the initial value for L3–L4, L4–L5, and L5–L6, respectively, within the first 10 minutes of recovery, then decreased. During the remaining 6 hours of the rest period, the mean NIEMG values of the multifidus muscles gradually increased to 139%, 143%, and 176% of their initial values for L3–L4, L4–L5, and L5–L6, respectively.

The mean NIEMG values of the multifidus muscles of the four preparations subjected to the 50 N load (Figure 4, bottom left) demonstrate an exponential decrease for the three lumbar levels for the 20-minute loading period, reaching 45%, 25%, and 46%, of their initial values. Spasms were observed in each preparation in the loading period. There was a transitory hyperexcitability lasting until the end of the first hour of recovery for L3–L4, L4–L5, and L5–L6 lumbar levels. At the end of the first 10 minutes of the recovery period, the mean NIEMG values peaked at 147%, 175%, and 354% of the initial value for L3–L4, L4–L5, and L5–L6 lumbar levels, respectively. The mean NIEMG values of the multifidus muscles then decreased, followed by gradual increase to 180% of its initial value for L4–L5, and to 196% and 202% of its initial value for L3–L4 and L5–L6 lumbar levels, respectively, at the end of the 7-hour recovery period.

The mean NIEMG values of the multifidus muscles of the six preparations subjected to the 70 N load (Figure 4, bottom right) demonstrate an exponential decrease for the three lumbar levels during the 20-minute loading period. For L3–L4, L4–L5, and L5–L6, the mean NIEMG values of the multifidus muscles decreased to 38%, 32%, and 42%, respectively, of their initial values. Spasms were present in each preparation during the 20 minutes. In the recovery period, there was a transitory hyperex-

Table 2. Mean Displacement Values Throughout the Experiment

Load N	Mean Initial Displacement	Mean Displacement at End of 20 Mins	Mean Displacement at End of 7 Hrs
20 N	7.18 (± 0.12) mm	12.1 (± 1.43) mm (+ 68.5%)	9.76 (± 1.37) mm (+ 35.9%)
30 N	9.24 (± 1.39) mm	15.5 (± 2.66) mm (+ 67.7%)	11.1 (± 1.25) mm (+ 20.2%)
50 N	13.2 (± 2.61) mm	20.8 (± 2.66) mm (+ 57.4%)	16.6 (± 2.02) mm (+ 25.8%)
70 N	16 (± 3.15) mm	26.5 (± 3.22) mm (+ 65.5%)	21.2 (± 3.18) mm (+ 32.4%)

Mean initial displacement = displacement immediately when full load was reached; mean displacement at 20 mins = the displacement just before load was removed; mean displacement at 7 hrs = the final value of the displacement after 7 hrs rest.

Table 3. Vertical Displacement Model During Load

$DISP(t) = D_0 + D_L(1 - e^{-t/T2})$

Load	D_0	D_L (mm)	T2 (mins)	r^2
20 N	8.426	3.430	3.587	0.9096
30 N	10.15	5.193	3.925	0.9734
50 N	14.49	5.991	4.163	0.9619
70 N	17.65	8.225	3.225	0.9552

Table 5. Vertical Displacement Model During Recovery

$DISP(t) = D_c + D_r e^{-t/T3}$

Load	D_c (mm)	D_r (mm)	T3 (mins)	r^2
20 N	10.12	1.965	8.361	0.9178
30 N	11.94	2.920	40.984	0.8022
50 N	17.15	3.594	5.851	0.8623
70 N	21.67	4.77	5.461	0.9629

citability in the first hour of recovery for each of the lumbar levels. At the end of the first 10 minutes of the recovery period, the mean NIEMG values peaked at 109%, 122%, and 108% for L3–L4, L4–L5, and L5–L6, respectively. At the end of the 7-hour recovery, the mean NIEMG values of the multifidus muscles increased to 133%, 139%, and 128% of their initial values for L3–L4, L4–L5, and L5–L6, respectively.

Model Development

Parameters for all models fitted were obtained by using the Marquardt-Levenberg nonlinear regression algorithm. The parameters for the vertical displacement model, as defined in equation (4), are shown in Table 3 and Figure 4. Within the 20 minutes of this test, only the fast decaying exponential component, which attributed to ligamentous viscoelasticity, manifested itself.

As defined in equation (3), the pooled NIEMG values for the 20 minutes of static loading took a decaying exponential form. The model parameters that produced the best model fit are shown in Table 4 and Figure 4 superimposed on the experimental data. The models for 20 N, 30 N, and 50 N of static loading showed similar behavior, in which the exponential decay rates increased with the higher amounts of load. For higher magnitudes of load, it took longer to achieve steady state. Whereas the lumbar levels that were far away from the load application point (L3–L4 and L5–L6) showed a slower behavior at higher NIEMG values, at the load application point (L4–L5) the NIEMG values reached the lowest levels with the highest decay rate. In the case of L5–L6 at 50 N, the model parameters were out of norm with the others, likely because of larger deviation from the norm caused by frequent and severe occurrences of spasms. This resulted in a loss of the exponential decaying nature of the other curves and decrease in the quality of model fit.

Table 4. Normalized EMG Model During Load

$NIEMG(t) = Ae^{-t/T1} + NIEMG_0$

Load	Level	$NIEMG_0$	A	T1 (mins)	r^2
20 N	L3–L4	0.6889	0.2881	0.992	0.9752
	L4–L5	0.6517	0.3897	2.191	0.9557
	L5–L6	0.7354	0.2606	0.778	0.9049
30 N	L3–L4	0.3498	0.6084	4.695	0.9435
	L4–L5	0.3554	0.5682	4.845	0.9794
	L5–L6	0.3421	0.5507	3.203	0.9409
50 N	L3–L4	0.4653	0.7050	5.139	0.7889
	L4–L5	0.2696	0.7982	3.676	0.9723
	L5–L6	0.0	1.019	32.154	0.4649
70 N	L3–L4	0.4158	0.5732	3.494	0.9865
	L4–L5	0.3818	0.5576	2.693	0.9616
	L5–L6	0.4731	0.5053	2.455	0.9787

The parameters for displacement recovery models are tabulated in Table 5 and shown in Figure 5. The recovery components tended to increase with increasing load, as did the amount of recoverable strain, represented by the column under parameter D_R in Table 5.

A relatively complex model was necessary in order to fit and explain physiologically the recovery behavior of NIEMG. The normal exponential recovery components had a magnitude directly related to the amount of desensitization occurring during load; physiologically, after some indefinite period of sufficient rest, spinal sensitivity to ligament strain may return to normal; mathematically, this is guaranteed by the modeling constraint that $E + NIEMG_0 = 1$. The time constant for this component is based on earlier work and was constrained to last between 4 and 8 hours.[32] The transient hyperexcitability seen in this response was also similar to what was observed in earlier work,[26] with time constant in the order of 6–16 minutes. Compounding this is a delayed hyperexcitability of long duration, with a time constant in the order of several hours, termed "morning after" behavior, akin to the delayed soreness and stiffness associated with prolonged or cyclical bouts of lumbar flexion. Its onset delay appeared to depend on load (appearing earlier at high loads), with the time constant also in the order of 4–11 hours. Table 6 provides the model parameters, and its graphical representation is given in Figure 5.

■ Discussion

Several important findings resulted from this investigation. Even a short period of static lumbar flexion causes a complex and relatively prolonged transient neuromuscular disorder that seems to be independent of the magnitude of the load sustained by the viscoelastic structures. The source of the neuromuscular disorder observed in this investigation seems to arise from the response of afferents to the creep developed in the viscoelastic tissues of the spine. Furthermore, the creep developed in the tissues from only 20 minutes of light constant load does not fully recover even after 7 hours of rest, leaving the spine vulnerable to cumulative injury.

The complex neuromuscular disorder elicited by a short period of 20 minutes of sustained static load is composed of four distinct components, two of which are

Table 6. NIEMG Models During Recovery

$$NIEMG(t) = E(1 - e^{-t/T4}) + tBe^{-t/T5} + C(t - Td)e^{-(t - Td)/T6} + NIEMG_0$$

		E	T4 (mins)	B	T5 (mins)	C	T6 (mins)	Td (mins)	$NIEMG_0$	r^2
20 N	L3–L4	0.3219	480.0	0.1596	6.834	0.0084	260.1	223.9	0.6781	0.9960
	L4–L5	0.2974	240.0	0.05052	11.81	0.0086	432.3	157.2	0.7026	0.9870
	L5–L6	0.3219	480.0	0.1596	6.834	0.0076	324.0	222.0	0.6781	0.9951
30 N	L3–L4	0.5064	240.0	0.06612	11.97	0.0114	143.2	147.4	0.4936	0.9648
	L4–L5	0.6181	240.0	0.1306	12.67	0.00356	391.3	21.92	0.3819	0.9805
	L5–L6	0.5799	240.0	0.1499	11.69	0.0037	666.7	0	0.4201	0.9945
01603	L3–L4	0.5017	240.0	0.2064	11.77	0.0056	515.6	337.0	0.4983	0.9749
	L4–L5	0.7111	240.0	0.3087	11.77	0.0099	263.3	0	0.2889	0.9854
	L5–L6	0.3913	240.0	0.6065	11.88	0.02557	210.6	0	0.6087	0.9157
70 N	L3–L4	0.5991	240.0	0.1335	13.42	0.00255	412.9	0	0.4009	0.9732
	L4–L5	0.6594	240.0	0.1732	13.43	0.00259	498.3	0	0.3406	0.9891
	L5–L6	0.5488	240.0	0.1071	16.44	0.00476	237.4	60.58	0.4512	0.9805

prominent in the postloading period. The four components are present in the loading and recovery period regardless of the magnitude of the applied load.

The first component of the neuromuscular disorder elicited by the static flexion consists of the exponential decay in EMG over time as described by the term:

$$Ae^{-t/T1}$$

As the flexion persists, the forces generated by the muscles decrease, reducing the stiffness of the spine and, thereby, its stability. For the constant load of 20 N, the NIEMG at the end of 20 minutes of flexion ranged between 61% and 71% of the initial NIEMG in the L3–L4 to L5–L6 vertebrae. Similarly, for 30 N, 50 N, and 70 N loads, the NIEMG at the end of the 20-minute static flexion decreased to 37–42%, 25–46%, and 32–42% of the initial NIEMG, respectively. The physiologic processes responsible for the decrease in muscle activity are multiple and complex. Afferents are classified as fast-adapting or slow-adapting. The fast-adapting afferents exhibit a large neural discharge on the application of a static mechanical stimulus to the tissues in which they are embedded. The discharge, however, diminishes within a short period followed by silence, although the stimulus is still applied. Slow-adapting receptors respond differently. The application of a stimulus results in a sharp rise in their neural discharge, which exhibits a moderate decrease after the stimulus arrives to its steady state. The neural discharge continues at a steady rate thereafter, as long as the stimulus is applied.

Considering that the afferents in the lumbar viscoelastic tissues consist of fast- and slow-adapting receptors and that the stimulus consists of a constant load and ongoing creep, the EMG response is not surprising. *Via* the spinal reflex pathways, the afferents excite the muscles on the application of the flexion, and as the initial (phasic) component settles the EMG decreases to a level corresponding to the static discharge of the slow-adapting receptors, and remains at that level as long as the load is continued.

The second component of the disorder consists of the spasms that were evident throughout the 20 minutes of sustained flexion in all the preparations tested. The spasms appeared in a random, unpredictable manner with respect to their timing, intensity, duration, and frequency. Spasms and elevated activity of posterior lumbar muscles have been electromyographically confirmed in patients with idiopathic and pathologic low back pain.[33–38] Invariably, spasms are a direct result of some damage to the different tissues of the spine. Pedersen *et al*[18] were the first to demonstrate that crushing of the supraspinous ligament in a feline preparation elicits spasms in the gluteus muscle, scientifically establishing the relationships of ligamentous tissue damage and muscle spasms. Tissue damage is also always associated with pain. Incorporating the different fragments of information presented above suggests that the chain of events observed in this study consisted of viscoelastic tissue damage–pain–spasms.

The ligamentous tissues of lumbar spine were, however, loaded and stretched within their physiologic range of 25–40%.[27,30] Yet tissue damage did occur. With 20 minutes of creep, the supraspinous ligament length increased by 6–22% (Table 1) of its resting length, and after 7 hours of rest it was still between 4% and 7% longer. It seems that the nervous system recognizes the onset of a long-lasting viscoelastic strain as tissue damage. Indeed, prolonged or repetitive exposure of tendons or ligaments to loads well below the physiologic limits result in microtrauma to the basic structure of the collagen fibers,[39,40] and in inflammatory response. Such microtrauma resembles a low-grade "sprain" response such as pain, spasms, and inflammatory signs.[41] This type of low-grade "sprain" may last for several days and is accompanied by pain and spasms. The fact that the initial rate at which the spine was loaded (see *Protocol*) was deliberately set to be slow enough to prevent rate-dependent damage[42] further supports the creep-dependent "sprain" theory. As the creep in the viscoelastic structures progressed over time with the sustained load, the tissues lost their functional properties, which probably excites the pain receptors within and triggers the pain and associated spasms. Bare nerve endings, which are the pain-mediating receptors, have been

shown to exist in the spinal ligaments, discs, and capsules.[17–19] Overall, this process may explain one type of "idiopathic low back pain," as the viscoelastic tissues were strained within their physiologic limits and with absence of other pathology to any spinal structure.

The third component of the neuromuscular disorder consists of the transient hyperexcitability of the muscles in the first hour of the recovery period, which is described by the term:

$$tBe^{-t/T5}$$

The peak of the hyperexcitability was observed within the first 10 minutes of rest (after the static load was removed), and the model suggests that it probably decayed completely within an hour after the peak. This transient hyperexcitability further supports the ligamentous "sprain" theory. Assuming that a microdamage was present in the viscoelastic tissues, any attempt to stretch them again, on flexion, triggered the pain receptors within and reflexively recruited higher than normal muscle forces to protect the injured tissues from the load and further damage. As soon as the viscoelastic structures recovered sufficiently (an hour later) and their functional properties were somewhat restored (as evident from the rising exponential EMG component

$$[(A(1 - e^{-t/T4})]),$$

the tissues were able to sustain the load applied while reflexively activating the muscle (*via* mechanoreceptors, not pain receptors) for sufficient support to prevent further aggravation of the damage. The initial hyperexcitability was probably conveyed by pain receptors attempting to increase the stiffness of the spine by the musculature, whereas an hour later, the tissues recover enough to activate the muscles *via* the non–pain-sensitive afferents (*e.g.*, Pacinian, Ruffini, and Golgi) to create the necessary spinal stiffness.

The fourth component of the neuromuscular disorder consists of delayed hyperexcitability, which is described by the term:

$$C(t - Td)e^{-(t - Td)/T6}$$

The time delay indicates that at some time, 1–3 hours after the rest period was initiated, a physiologic–metabolic process (*e.g.*, most likely inflammation) began and reached a peak between 6 and 9 hours later. The peak amplitude of the delayed NIEMG activity was, in all cases, above the peak NIEMG recorded at the beginning of the 20-minute loading period. In some cases, it was 2–3 times of the initial NIEMG (Figure 5), indicating that this hyperexcitability is a significant disorder. Because the data were collected during a 7-hour rest period after the 20-minute loading, it was not possible to observe the exact time at which this hyperexcitability reached its peak in all cases. In some preparations the hyperexcitability peaked in the 6th hour and then slightly decreased, suggesting that the process matured. The model also points out that, in most cases, the peak of the hyperexcitability ranges from 7 to 9 hours and that it will decay to 5% of its peak after 24 hours. In most cases of subacute damage of viscoelastic tissues, pain, edema, and muscle stiffness (indicating the presence of inflammation[41]) are most pronounced in the "morning after" the injury. The same inflammatory process may take place in this case as well, giving rise to the delayed hyperexcitability several hours after the damage was inflicted. Ongoing work in our laboratory confirms the development of inflammation after the second hour.

The reality of the four components of neuromuscular disorder is familiar to nearly every individual who was engaged in weekend gardening or working on his automobile engine. Sustained static flexion during such activities eventually results in extreme discomfort or pain that is substantially relieved on extension. The next round of discomfort/pain in the posterior muscles is felt as the individual attempts to perform flexion to collect tools left on the floor. These sensations are mostly dormant during the rest of the day if additional work is not attempted. The next round of discomfort/pain and muscle stiffness is felt in the "morning after," following 7 to 8 hours of sleep. The delayed hyperexcitability of the muscles, termed the "morning after" hyperexcitability, produces the discomfort/pain and stiffness one feels in the days after performing the sustained static flexion. This disorder may last 1–3 days following which the episode is forgotten. The discomfort/pain during the sustained flexion is suggested by the spasms observed during the 20 minutes of loading. The discomfort/pain on performing flexion immediately after static activity is suggested by the initial hyperexcitability in the first hour of recovery in our model. The discomfort/pain felt in the morning after is probably the EMG manifestation of the inflammatory effects. The exponential decrease in muscle activity during the static flexion is probably not noticed by most individuals, although it may increase the exposure to injury. The graphical depiction of the four components of the disorder is shown in Figure 6. Each of the components, except the spasms, is described mathematically by the model developed earlier.

The data generated in this investigation shed some additional light on the controversy surrounding the utility and meaning of load control *versus* displacement control in physiologic research. In our previous reports,[26,27] similar preparations were subjected to static constant displacement *via* the L4–L5 supraspinous ligament as well as to 7 hours of recovery. The common features to the present and previous studies are the exponential decay of the EMG during the sustained flexion, the spasm that appeared in that period, and the initial hyperexcitability in the first hour of recovery. Both conditions also display an exponential increase of EMG during the recovery period. The major difference between the outcome of displacement control *versus* load control is the "morning after" hyperexcitability seen only in load control. The load control mode assures that the same load is

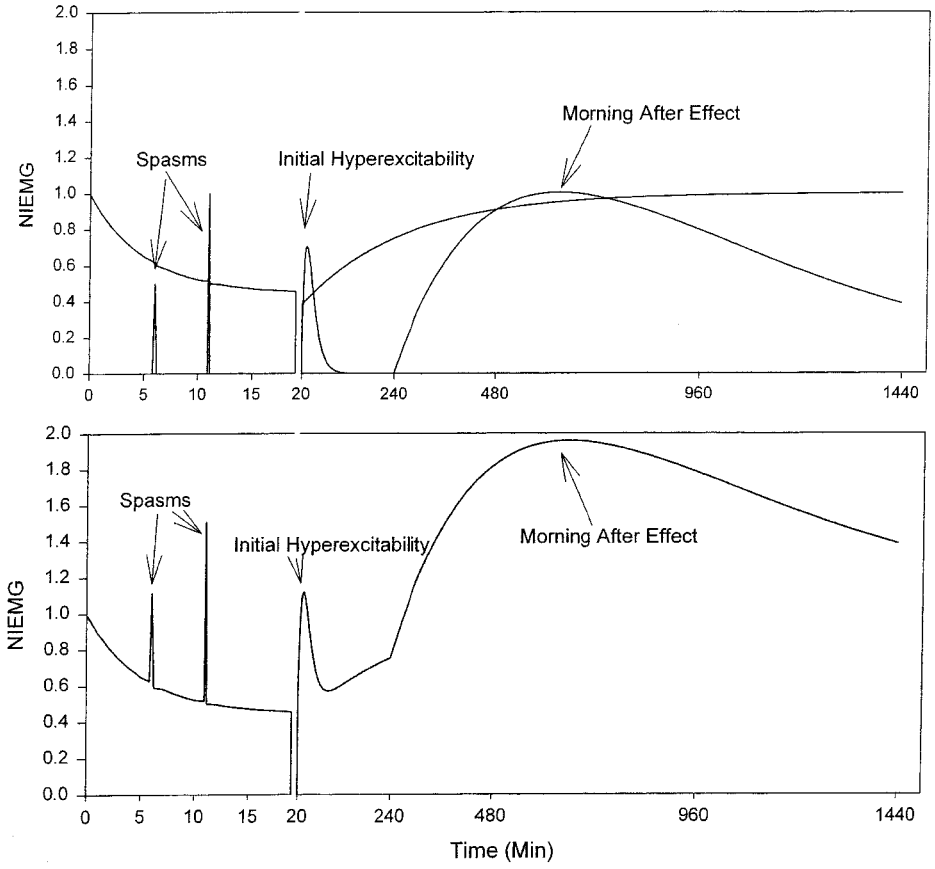

Figure 6. (Top) Schematic of the four components of the neuromuscular disorder resulting from the sustained static flexion: the decrease in muscular forces throughout the flexion, the spasms elicited by the microtrauma in the collagen fibers of the viscoelastic structures, the initial muscular hyperexcitability immediately after the static flexion, and the "morning after" excitability resulting by the inflammation developed in the viscoelastic tissues. (Bottom) The addition of all the components to result in a flexion and recovery response similar to that obtained in the experimental data.

applied to the spine despite the ongoing development of creep in the various viscoelastic tissues. In the ligaments, load control causes the tissue to creep (stretch) under constant load throughout the exposure period, resulting in significant elongation. Under displacement control, the ligament will develop initial tension while stretching. Once it stretches, the tension decreases (*e.g.*, tension–relaxation) exponentially, causing progressively lower challenge to the tissue. Overall, load control subjects the tissue to significantly higher strain than that in displacement control. The larger strain associated with load control probably causes a more severe subacute damage (*e.g.*, low-grade sprain) to the ligament, which results in inflammation and the delayed hyperexcitability ("morning after").

Indeed, under displacement control, the mean length of the L4–L5 supraspinous ligament at the end of 20-minute flexion was 7.15% longer than its initial length at the 1 N test load.[26,27] Under the constant load mode in the present study, the L4–L5 supraspinous length tested at the 1 N load at the end of the 20-minute flexion was 6.01% for the 20 N load trials and 12.37%, 13.1%, and 22.5% for the 30 N, 50 N, and 70 N loads, respectively. With the exception of the strain at the 20 N load, the values were 2–3 times that of the 7.15% strain developed in the constant displacement mode. The constant load, therefore, was a much more severe stimulus to the viscoelastic structures from the standpoint of creep developed during a 20-minute static flexion.

Another component of this study varied substantially from that of the constant displacement mode we investigated earlier.[26] The EMG recorded during the 20 minutes of static flexion decayed to 5% of its initial value in the constant displacement mode, whereas it decayed only to 32% to 71% of its initial values (depending on load magnitude) in the constant load mode used in this investigation. The EMG at the end of the 20-minute test in the constant load mode was six or more times that in the displacement mode. In the displacement mode, the decrease to the 5% of the initial value occurred within the first 5 minutes, whereas in the constant load mode the values given above were at the very end of the 20-minute session.

In reality, spine functions during the performance of routine daily personal, occupational, and sports activity are composed of neither pure constant displacement nor constant load. Spinal motion is probably a combination of load and displacement control. A laborer may lift boxes and load them on a cart or truck. The spine has to flex or extend from point A (initial location of the box) to point B (final location of the box) but has to sustain the constant mass of the box. The actual load will change with the acceleration and deceleration associated with the movement,[29,31,43] the orientation of the gravity vector, and the moment arm with respect to rotational centers. In more static conditions, a concrete fabricator may be flexed to reach a fixed angle, and at the same time he has to support the load of his tools as well as the weight

of his upper torso, a combination of constant displacement and constant load.

It is unlikely that any one real-life activity is represented exclusively by load control or displacement control. More likely, real-life activities are complex combinations of both. The combination of simultaneous load and displacement control in real-life activities may represent a much more challenging condition to the viscoelastic tissues than the separate impact of each of the two paradigms. Yet, each one of the two test paradigms provides a unique perspective into spinal function/dysfunction. Better yet, both paradigms should be used to enrich our knowledge and experience and expand the insights into spine function/dysfunction as was found in this study.

The neuromuscular disorder composed of the four components was not dependent on the magnitude of the load applied. This was somewhat of a surprise for the ligament load of 20 N. It was expected that such a mild load will not cause substantial creep and the associated subacute damage and inflammation as would a heavier load. The four components of the disorder were, however, fully present in the test at 20 N. Hagg observed disorganized spasms in the hand/wrist and shoulder muscles of workers performing static work over a prolonged period.[44,45] In his work, the muscular effort was as low as 5% of the maximal voluntary force available from the muscles. The data we obtained tend to support his findings and to also support that static loads, even at low magnitudes, are a risk factor for MSDs. However, his subjects were healthy humans under voluntary control, whereas feline specimen subjected to passive loads were used in this report. Furthermore, the fact that such a disorder was so far observed in the spine, shoulder, and hand/wrist suggests that a similar phenomenon may be present in other joints when exposed to prolonged static loading condition.

The pattern of EMG activity observed in this study suggests that the physiology and biomechanics of a common idiopathic low back pain are at hand. Often, patients complaining of low back pain are examined in the clinic, and the diagnostic tests do not show any pathology (such as prolapsed disc, impingement, and fracture). Most caregivers prescribe up to 2 weeks of rest with or without pain medication or muscle relaxants if spasms are evident. In many cases, the problem is resolved within a few days, and the patient returns to routine daily activities. Based on the results of this study, anti-inflammatory medication may be better suited.

The neuromuscular disorder described in this report was elicited by 20 minutes of sustained flexion followed by 7 hours of rest. The model also predicts implications of the disorder up to 24 hours past the sustained flexion. Although such a disorder is transient, its implications on individuals who are subjected to daily work over weeks and months is far from being transient. Evidently, workers such as farm hands, concrete fabricators, mechanics, and brick/floor/carpet layers, perform repeated bouts of sustained flexion over a day's work. This exposes the spine to the cumulative effects of viscoelastic creep, which is not fully recovered from one bout of flexion to the next. Over several hours the creep increases to a magnitude that surely will not recover until the next workday. The worker begins another day of sustained flexion periods with residual creep in the tissues from the day before. Over time, the accumulated creep increases and the viscoelastic tissues may be subjected to permanent loss of their ability to recover to their original resting length. Furthermore, the inflammatory process may become chronic and present a serious medical challenge to negotiate. Indeed, Safran[41] documented that if the rate of microtrauma healing does not exceed the rate of microtrauma production, a chronic inflammation and disability of the patient may result. The findings presented here, therefore, may be the physiologic and biomechanical infrastructure for the explanation of cumulative trauma disorders.

■ Conclusion

Based on the results of this study, the following conclusions are offered: static lumbar flexion under constant load results in long-lasting viscoelastic creep that does not fully recover after 7 hours of rest. The creep developed gives rise to a neuromuscular disorder composed of reduced reflexive muscle activity and superimposed spasms during flexion and two types of hyperexcitability during a following rest period, one of which may last well beyond 24 hours. Viscoelastic creep and the associated neuromuscular disorder are relatively independent of the static load magnitude: *e.g.*, even low loads applied in static flexion elicit a disorder. Although many types of idiopathic low back disorders exist, the work presented here provides a biomechanical and electromyographic infrastructure that could explain one of the most common of this class of disorders and an insight on how cumulative low back problems may develop.

■ Key Points

- Static lumbar flexion results in creep of viscoelastic tissues, microdamage, and a neuromuscular disorder.
- The development of the neuromuscular disorder is relatively independent of the magnitude of the load applied.
- The disorder is elicited even when the viscoelastic tissues are strained within their physiologic range.

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