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Multifidus EMG and Tension–Relaxation Recovery After Prolonged Static Lumbar Flexion

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Study Design. The electromyogram (EMG) from the *in vivo* feline L1 to the L7 multifidus was recorded during the application of a 20-minute static lumbar flexion and after 7 hours of rest.

Objective. To determine the recovery of tension–relaxation and laxity in the lumbar viscoelastic structures as well as the recovery of reflexive EMG activity in the multifidus after prolonged static flexion.

Summary of Background. It has been established that prolonged static flexion of the spine induces creep or tension–relaxation in its viscoelastic structures as well as a sharp decrease in the reflexive activity of the dorsal musculature and initiation of spasms. Epidemiologic studies have pointed out that such static flexion is associated with unusually high rates of low back disorders. The rate and pattern of recovery of reflexive muscular activity with rest after static flexion is still unknown.

Methods. The lumbar spines of seven *in vivo* feline preparations were subjected to 20 minutes of passive anterior flexion followed by 7 hours of rest while monitoring flexion tension, EMG from the L1–L7 multifidus muscles, and the strain of the L4/L5 supraspinal ligament. A model describing the pattern of recovery of muscular activity and viscoelastic tension was developed.

Results. Twenty minutes of lumbar flexion was associated with an initial sharp decrease of multifidus EMG activity followed by spasms. During rest, EMG activity demonstrated an initial hyperexcitability on flexion, followed by an exponential recovery of muscle activity. Full recovery of residual strain in the L4/L5 supraspinous ligament and multifidus activity was not obtained after 7 hours of rest.

Conclusions. Static flexion of the lumbar spine is an extremely imposing function on its viscoelastic tissues, resulting in spasms and requiring long periods of rest before normal functions are re-established. [Key words: EMG, ergonomics, lumbar, multifidus, spasms, spine] **Spine 2001;26:715–723**

Occupational and sports activities requiring cyclic or static lumbar flexion are associated with unusually high episodes of low back pain and injury.[3,17,22,23,28] Recent evidence has demonstrated that cyclic flexion activities give rise to tension–relaxation or creep of viscoelastic structures[1,2,6,10,13,16,19,32] and to a sharp decline of reflexive activation of the musclature[32,36] known to stabilize the spine.[8,18,25,26,35,39] Full recovery of such reflexive activity may require up to 8 hours of rest.[7,33] Meanwhile, the spine may be exposed to motion without the natural protective stiffness afforded by the musculature, increasing the possibility of instability, injury, and pain.

Recent data also has shown that prolonged static flexion of the spine is accompanied by tension–relaxation of its viscoelastic tissue and with a sharp initial decrease in the reflexive activity level of the multifidus followed by EMG spasms of various intensities appearing at random and at a totally unpredictable frequency.[24,36] The pattern and length of the recovery period necessary to restore normal physiologic functions of the viscoelastic structures and muscular activity after static flexion, however, are still unknown.

The objectives of this investigation, therefore, were to assess the duration and pattern of the recovery of the multifidus muscles' reflexive activation as well as recovery of the tension–relaxation of the lumbar viscoelastic tissues after a period of static lumbar flexion. The information derived from such research may give new insights into spinal mechanics and disorders as well as into optimal design of work/rest periods in occupational and sports activities.

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Methods

Preparation. Seven adult cats (4.71 ± 0.37 kg) were anesthetized with a single injection of chloralose (60 mg/kg) in a protocol approved by the Institutional Animal Care and Use Committee (IACUC). A booster injection was given whenever the depth of anesthesia was insufficient as judged by testing eye reflexes. 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. The preparation was placed in a rigid stainless steel frame that allowed the isolation of various lumbar levels by external fixation. A gauze pad soaked with saline was applied over the incision during the experiment to prevent the exposed tissue from drying.

Instrumentation. Six pairs of stainless steel fine wire EMG electrodes, insulated except for a 1-mm exposed tip, were inserted through hypodermic needles into the multifidus muscles of the L1/L2, L2/L3 . . . L6/L7 (the cat has seven lumbar vertebrae) on the right side, 5–6 mm from the midline. The interelectrode distance of each pair was 3–4 mm. A ground electrode was inserted into 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 bandpass filter of 6–500 Hz. EMG response from each

channel was monitored on oscilloscopes and stored in the computer, with a sampling rate of 1000 Hz.

An "S"-shaped stainless steel hook was inserted around the middle part of the L4/L5 supraspinous ligament and connected to the vertical actuator of a Bionic 858 Material Testing System (MTS, Inc., Minneapolis, MN) instrumented with a computer-controlled loading system. The load cell output of the Bionic 858 was sampled into the computer along with the EMG data.

The lumbar spine was isolated by applying one external fixator to the L1 posterior spinal process and a second fixator to the L7 process. The external fixation was not intended to prevent micromotion or macromotion of the vertebrae, but to limit the elicited flexion to the lumbar spine and to prevent interaction of thoracic and sacral/pelvic structures.

Protocol. The stainless steel hook applied to the L4/L5 supraspinous ligament was pulled up by the Bionic 858 system while controlling a step-and-hold displacement of 12–15 mm (according to the specimen size) from a resting position with a 0.5-N preload applied just before the step and hold. The 12–15 mm displacement was calculated and tested to provide mostly an axial strain in the supraspinous ligament within its physiologic range (See Appendix A in Reference 36 for details) in the initial phase of the displacement (0–5 mm) and then displace the adjacent lumbar vertebrae into moderate flexion (5–15 mm) such that viscoelastic structures (various ligaments, discs, and facet capsules) of several lumbar motion segments were deformed as well.

The step-and-hold displacement was applied for a period of 20 minutes, after which it was returned to the original baseline (*e.g.*, zero displacement at rest). After the return to zero displacement, the preparation was allowed to recover at rest for 7 hours, during which short 6-second tests (2 seconds ramp, 2 seconds at full displacement, and 2 seconds ramp down) at full original displacement (12–15 mm) were applied to assess recovery of tension in the ligament/spine and EMG in the six lumbar levels. The timing of the test application was at 10 minutes, 30 minutes, and 60 minutes after initiation of the rest period and every hour thereafter up to 7 hours.

Using two short hypodermic needles inserted into the spinal processes of L4 and L5 (See Appendix A in Reference 36), the length of the supraspinous ligament of that segment was measured with calipers while the 0.5 N preload was applied just before the onset of the 20-minute flexion session. Immediately after the 20-minute step-and-hold displacement was returned to zero, the displacement was reset to yield 0.5 N (to offset the laxity developed in the ligament during the 20-minute session) while recording the vertical displacement of the load cell required to elicit the 0.5-N load and the length of the L4/L5 supraspinous ligament. These measurements were used to estimate the residual axial strain in the ligament at the end of the 20-minute flexion and at the end of the 7 hours of recovery (details were given in Appendix B in Reference 36).

EMG readings from the six multifidus muscles and tension were recorded continuously during the 20-minute displacement period and during the 6-second tests applied in the recovery period.

Analysis. Two-second windows of EMG and tension that developed in the spine were extracted immediately at the beginning of the displacement period and every 20 seconds thereafter for the full 20 minutes. Each EMG sample during the initial 20 minutes and then recovery was full wave rectified and digitally integrated over the 2-second window. The value obtained from the integration at the end of each 2-second window was divided by the value obtained from the very first window, yielding a normalized value. The normalized integrated EMG (NIEMG) of all the preparations at the respective window were pooled, and the mean (± SD) was calculated and plotted on a NIEMG *versus* time plot for each of the multifidus muscles of the six levels.

Similarly, the tension recorded just at the beginning of the displacement period was used for normalization of the tension values sampled consequently. Normalized tension of the respective window of all preparations were pooled, and the mean (± SD) was calculated and plotted as a normalized load *versus* time plot.

Using the measurements of the supraspinous ligament length at 0.5-N preload before and immediately after the 20-minute displacement was applied and the vertical displacement of the load cell required to elicit 0.5-N load at the end of the 20-minute trial and at the end of the 7 hours recovery period, the residual axial strain in the ligament was calculated (as described in Appendix B given in Reference 36).

Model Development. The mean (± SD) NIEMG data recorded from the multifidus muscles of the six lumbar levels as well as the tension recorded in the load cell in the 2-second tests during the recovery period were fitted with a model consisting of three terms. Two exponential terms were chosen because they represent the classic behavior of viscoelastic structures such as ligaments, discs, *etc.*, as was shown by previous studies when cyclic flexion was applied.[7,24,33]

The recovery model, based on previous studies,[7,33] consists of three components: a residual, comprising the lowest level of force or EMG activity at the end of the flexion period; a fast asymptotic exponential recovery, and a slow, delayed asymptotic exponential recovery. This model is of the following format:

$$y(t) = A(1 - e^{-\frac{t}{T1}}) + B(1 - e^{-\frac{-(t-\tau)}{T2}}) + R \qquad (a)$$

where:

A is the amplitude of the fast recovery component (% initial value),

T1 is the time constant of the fast recovery component (minutes),

B is the amplitude of the slow recovery component (% initial value),

T2 is the time constant of the slow recovery component (minutes),

τ is the time delay of the slow recovery component (minutes), and

R is the residual component left at the end of the 20-minute flexion period (% initial value)

A Marquardt-Levenberg nonlinear algorithm was used to estimate the best-fitting model parameters.

■ Results

A typical response consisting of raw EMG and tension readings during the 20 minutes of static flexion and the consequent 7 hours of recovery is shown in Figure 1. The bottom trace shows the tension–relaxation of the supraspinous ligament of the L4/L5 segment as well as the

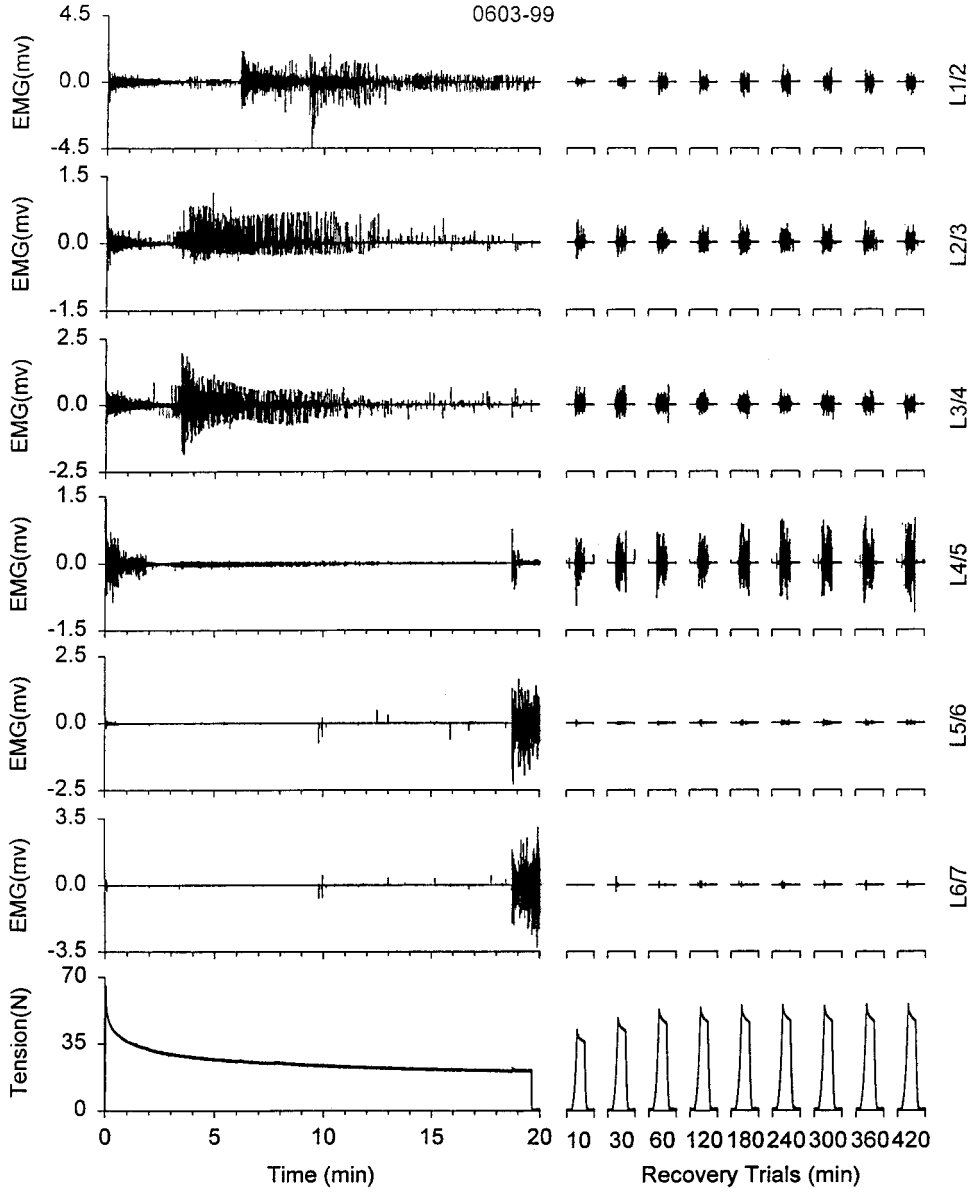

Figure 1. Typical raw EMG and tension responses during the static flexion of the lumbar spine, as well as during the recovery period.

other viscoelastic structures. The exponential decay at the beginning of the static flexion resulted in a 45% loss of the tension within the first minute and additional decrease during the following 5 minutes. The decrease in tension past the first 5 minutes was slower and asymptotically arrived to 32% of the initial tension at the end of the 20-minute test. After 10 minutes of rest, a substantial amount of recovery was present, and the recovery increased in an exponential fashion until the end of the 7-hour recovery period.

The EMG readings recorded from the six lumbar levels contained large initial discharge associated with the onset of the flexion in the lumbar spine. The EMG values also decreased in an exponential fashion in parallel with the tension–relaxation values exhibited by the spine, reaching 5% of its initial value within 3–4 minutes. Thereafter, large amplitude spasms were recorded. The spasms appeared at random throughout the static flexion period, without establishing a pattern in timing of appearance, duration, or intensity.

During the recovery period, the EMG readings exhibited large initial recovery, a minor decrease after 30 minutes of rest, and a slow exponential increase thereafter.

Figure 2 provides the mean (± SD) of the normalized integrated EMG (NIEMG) recorded from the six lumbar levels as well as the mean (± SD) tension recorded from the seven preparations used in this study. The mean normalized tension of the seven preparations decreased to 60% of its initial value within the first 20 seconds of the static flexion and continued to decrease further thereafter, reaching 41% of its initial value at the end of 5 minutes. The loss of tension was much slower thereafter, ending with 32% of its initial value at the end of the 20 minutes of flexion. Overall, the tension–relaxation demonstrated a loss of 68% during the 20 minutes of lumbar flexion.

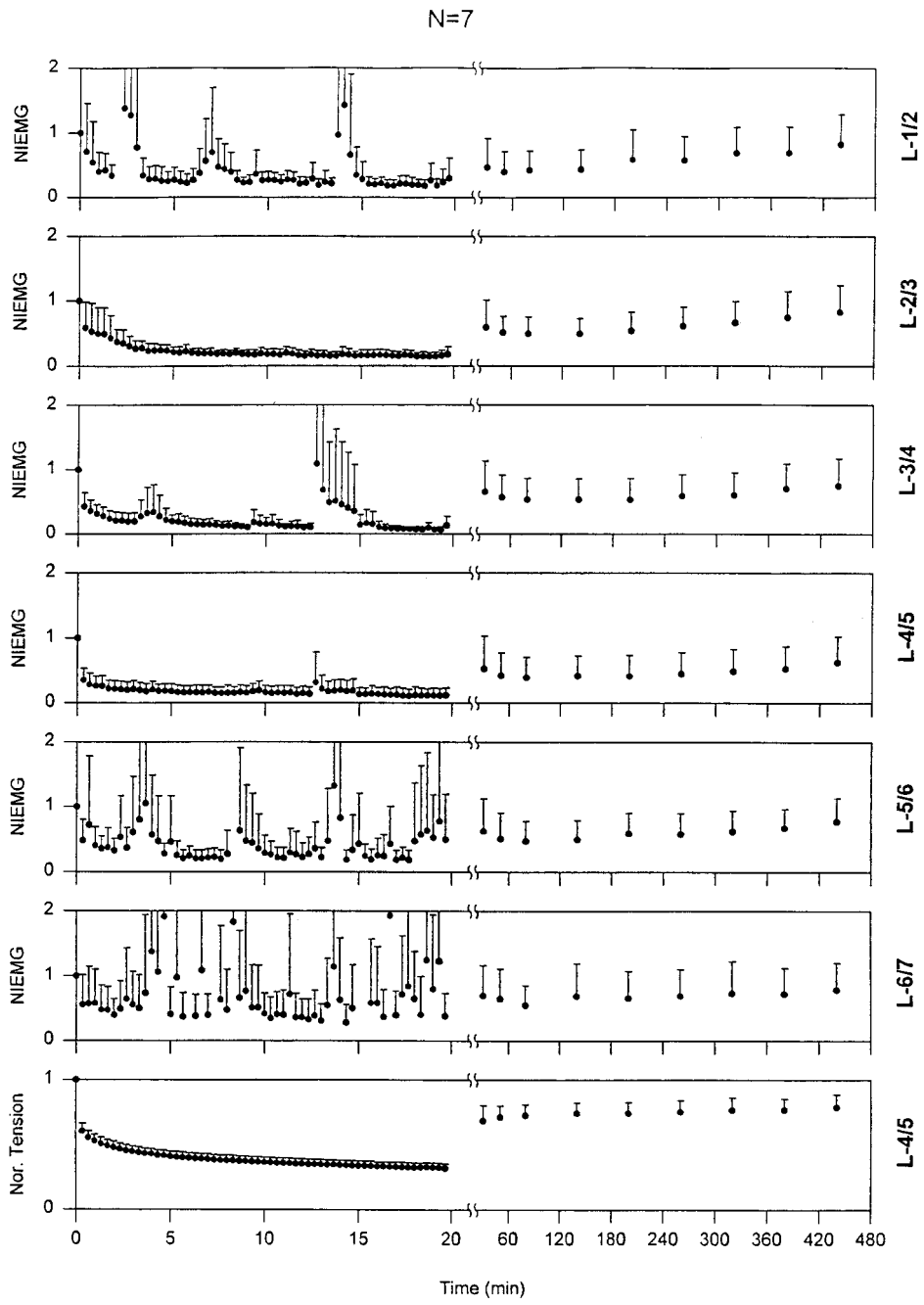

Figure 2. The mean (± SD) normalized integrated EMG and the mean (± SD) tension of the seven preparations. Note the exponential decline of the NIEMG that is interrupted by the spasms throughout the static flexion period. Also note that the NIEMG exhibits an initial fast recovery that peaks during the first recovery hour, slightly declines thereafter, and is followed by a slow gradual increase.

The first 10 minutes of rest were characterized by recovery of the tension from 32% to 68%, a 36% recovery. The recovery of the tension was very slow thereafter, arriving to 79% of its original value at the end of 7 hours of rest. Full recovery was not observed in any of the preparations. The mean NIEMG of the multifidus muscles of the six levels demonstrates an exponential decrease in the first 3–5 minutes from the onset of the 20 minutes of flexion and a slow decrease thereafter. Because the decrease was interrupted by EMG spasms that appeared at random in any one of the levels, the actual data demonstrated large variability, precluding it from a systematic analysis.

The recovery period, however, was consistent throughout the preparations, demonstrating increases of 17, 41, 53, 41, 13, and 32% in the multifidus of L1/L2, L2/L3 . . . L6/L7, respectively, after 10 minutes of rest. An additional 20 minutes of rest resulted in a consistent decrease of 5–10% in the NIEMG from each level. The NIEMG then gradually increased throughout the 7 hours of rest reaching 83, 82, 75, 63, 77, and 79% of the initial values of the multifidus of L1/L2 . . . L6/L7, respectively. Full recovery was observed in one or two levels of some preparations, but none of the mean data exhibited full recovery. Table 1 provides the means (± SD) of the NIEMG and tension data during the flexion and recovery periods.

The mean axial residual strain in the supraspinal ligament at the end of the 20 minutes of flexion was calculated to be 7.15 ± 2.11%, and the mean axial residual

Table 1. Mean and Standard Deviation of Normalized IEMG and Tension

	Time	NIEMG L1/L2	NIEMG L2/L3	NIEMG L3/L4	NIEMG L4/L5	NIEMG L5/L6	NIEMG L6/L7	Tension
Flexion								
	0 min	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00
	20 sec	0.71 ± 0.74	0.59 ± 0.40	0.43 ± 0.22	0.35 ± 0.18	0.48 ± 0.32	0.56 ± 0.46	0.60 ± 0.06
	40 sec	0.54 ± 0.63	0.53 ± 0.43	0.36 ± 0.17	0.28 ± 0.18	0.71 ± 1.07	0.57 ± 0.58	0.55 ± 0.05
	1 min	0.40 ± 0.30	0.49 ± 0.41	0.31 ± 0.14	0.26 ± 0.15	0.40 ± 0.29	0.57 ± 0.53	0.53 ± 0.05
	2 min	2.56 ± 6.07	0.37 ± 0.19	0.21 ± 0.14	0.21 ± 0.13	0.32 ± 0.18	0.39 ± 0.25	0.48 ± 0.05
	3 min	0.78 ± 1.36	0.27 ± 0.12	0.20 ± 0.13	0.21 ± 0.13	0.61 ± 0.86	0.55 ± 0.51	0.45 ± 0.04
	4 min	0.29 ± 0.21	0.24 ± 0.10	0.34 ± 0.43	0.21 ± 0.13	0.57 ± 0.92	1.37 ± 2.34	0.43 ± 0.04
	5 min	0.27 ± 0.19	0.21 ± 0.08	0.19 ± 0.10	0.18 ± 0.12	0.46 ± 0.71	0.41 ± 0.42	0.41 ± 0.04
	10 min	0.27 ± 0.13	0.18 ± 0.08	0.15 ± 0.10	0.16 ± 0.11	0.28 ± 0.28	0.41 ± 0.32	0.36 ± 0.04
	15 min	0.28 ± 0.28	0.17 ± 0.08	0.14 ± 0.16	0.13 ± 0.10	0.43 ± 0.78	2.32 ± 5.30	0.34 ± 0.04
	20 min	0.30 ± 0.31	0.18 ± 0.11	0.13 ± 0.14	0.12 ± 0.12	0.49 ± 0.70	0.38 ± 0.35	0.32 ± 0.03
Recovery								
	30 min	0.47 ± 0.45	0.59 ± 0.42	0.66 ± 0.48	0.53 ± 0.50	0.62 ± 0.50	0.70 ± 0.47	0.68 ± 0.12
	50 min	0.40 ± 0.32	0.51 ± 0.25	0.57 ± 0.35	0.42 ± 0.35	0.51 ± 0.40	0.65 ± 0.47	0.71 ± 0.09
	80 min	0.43 ± 0.30	0.49 ± 0.26	0.54 ± 0.33	0.39 ± 0.31	0.47 ± 0.31	0.55 ± 0.31	0.73 ± 0.08
	140 min	0.43 ± 0.31	0.49 ± 0.23	0.54 ± 0.32	0.42 ± 0.31	0.50 ± 0.29	0.69 ± 0.51	0.74 ± 0.08
	200 min	0.58 ± 0.46	0.54 ± 0.29	0.53 ± 0.33	0.41 ± 0.32	0.59 ± 0.31	0.66 ± 0.42	0.74 ± 0.09
	260 min	0.57 ± 0.37	0.61 ± 0.29	0.59 ± 0.33	0.45 ± 0.33	0.58 ± 0.32	0.69 ± 0.41	0.75 ± 0.09
	320 min	0.69 ± 0.40	0.66 ± 0.33	0.60 ± 0.35	0.49 ± 0.34	0.62 ± 0.32	0.74 ± 0.49	0.77 ± 0.10
	380 min	0.68 ± 0.40	0.74 ± 0.40	0.71 ± 0.38	0.53 ± 0.34	0.67 ± 0.29	0.72 ± 0.40	0.77 ± 0.09
	440 min	0.83 ± 0.46	0.82 ± 0.42	0.75 ± 0.42	0.63 ± 0.39	0.77 ± 0.37	0.79 ± 0.42	0.79 ± 0.10

NIEMG = normalized integrated EMG.

strain at the end of the 7-hour recovery period was 0.87 ± 0.26% (see Appendix B in Reference 36).

Figure 3 provides the best-fit model for the recovery of NIEMG and tension data. Because of the NIEMG transient hyperexcitability apparent in the first 30 minutes of recovery, a third component was added to the anticipated mathematical model shown earlier in Equation (a). The new term is shown below:

$$Cte^{-\frac{t}{T3}} \quad \text{(b)}$$

Where:

C is the amplitude of the transient hyperexcitability component (% initial value), and

T3 is the time constant of the transient hyperexcitability component (minutes)

Exponential terms for the model were selected *a priori*, because they provide the classic description of viscoelastic behavior. The transient term properly describes the temporary increase in EMG values in the first 30 minutes of the recovery period.

Parameters for the model were found through an iterative process as follows: First, the parameter R (value at start of recovery) was directly extracted as the final point of the tension or NIEMG at the end of the 20 minutes of flexion. Because the demarcation between first and second recovery components was clear, the first plateau level (minus the parameter R) was used for A. Next, the time constant T1 was estimated through iteration. Once the parameters for the first component were estimated, the second component was addressed. The EMG data showed that the initiation time for the second component was between the second and third hour of recovery; thus, a constraint was placed on this parameter to be between 120–180 minutes in the EMG models. Then, the maximal value of the total recovery (minus the parameters R and A) was used as B. Then, the time constant of the second recovery (T2) was estimated. Finally, for the NIEMG data, the parameters C and T3 were estimated by the amplitude and duration of the transient hyperexcitability. This component was not used for the tension fits, because the hyperexcitability was a purely neurophysiologic manifestation, with no evidence of such behavior in the tension data. These initial parameters were input to a Marquardt–Levenberg nonlinear regression algorithm for final iterations. The success of this process could be limited for two main reasons: first, the partial derivatives computed within the algorithm tended to overflow because of the use of saturated exponentials, and second, the parameters of the second component were highly dependent on each other and had large final coefficients of variation. Thus, once the model format was set, the fits were relatively insensitive to changes in parameters. It should be kept in mind that the parameters shown here are approximate in nature, reflecting the overall behavior of the recovery response, and not precise determinations of this highly variable phenomenon. The authors therefore opted for parameters to two significant digits, in the middle of each parameter's confidence interval.

Table 2 summarizes the final parameters and coefficients of the model for each of the multifidus levels (*e.g.*, L1/L2–L6/L7) and for the tension model. Again, in the tension model, the transient term was absent because it was not present in the experimental data. Squared correlation coefficients (r^2) were calculated as the ratio of the sum of squared deviation of the data points from their mean accounted for by the model divided by the

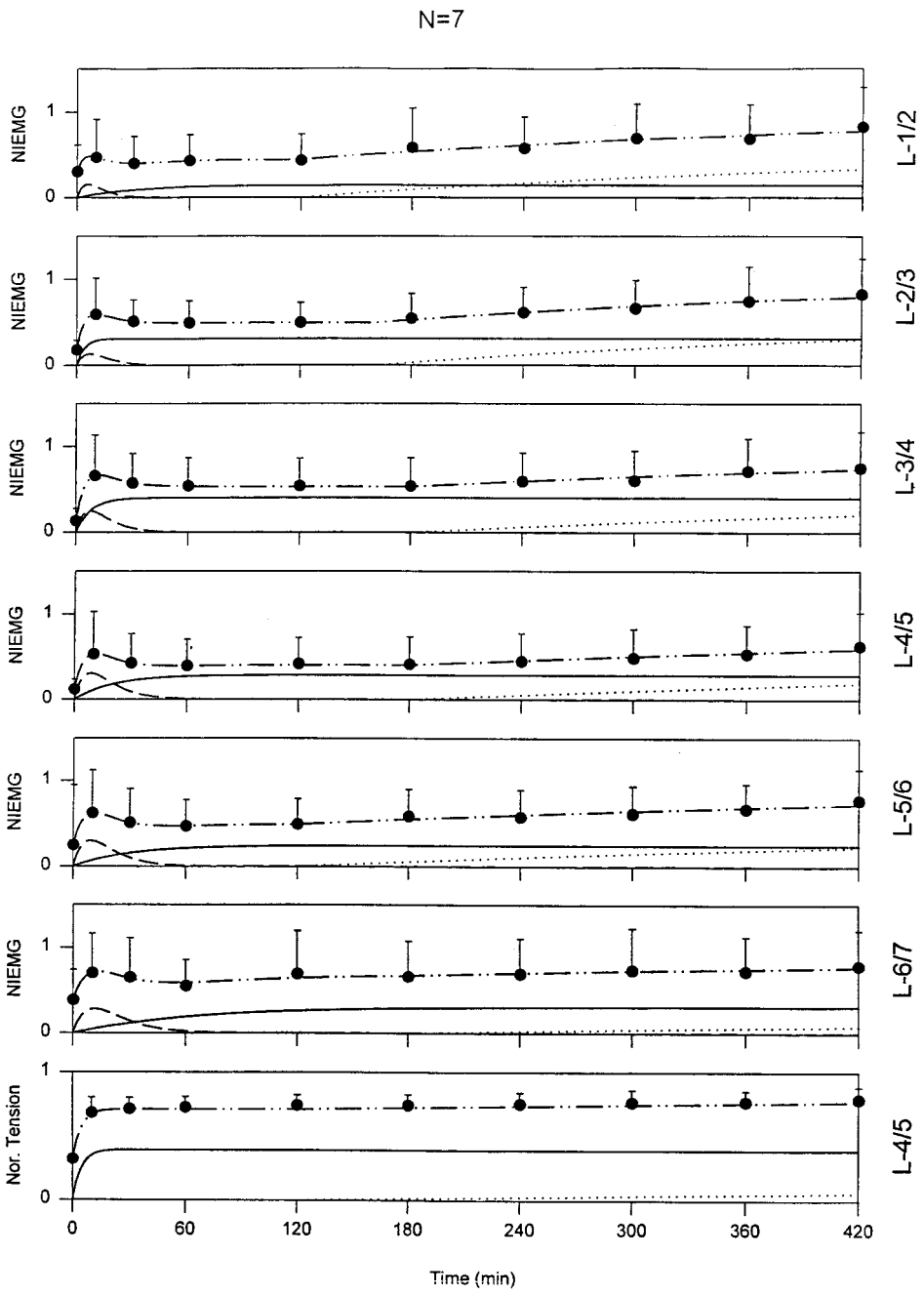

Figure 3. The best-fit model to the recovery data. The solid line depicts the fast recovery component, whereas the dotted line depicts the slow recovery component. The broken lines depict the transient hyperexcitability of the muscles in the first hour after the static flexion. The complete model (depicted by the broken line spaced by two dots) for multifidus muscles of each level and for the tension is the sum of all three components and the residual, and it goes through the experimental data points. The coefficients for the best model for each lumbar level and the tension is given in Table 2.

total sum of squared deviation of the data points from their mean.

■ Discussion

Several important issues emerge from the results of this investigation. The data confirm the previous observation that prolonged static flexion of the lumbar spine results in fast exponential decrease of reflexive muscular activity (which provides the stability of the spine) and the initiation of spasms that appear spontaneously and randomly.[36] The loss of muscular stabilizing forces follows the tension–relaxation and is accompanied by a residual

Table 2. Parameters Associated with the Tension and EMG Models of Each Lumbar Level During the Recovery Period

	A	T1	B	T2	τ	C	T3	R	r^2
L1/L2	0.14	31	0.55	330	120	0.076	5.6	0.30	0.962
L2/L3	0.31	3.9	0.51	290	160	0.053	6.9	0.18	0.993
L3/L4	0.40	7.8	0.47	420	180	0.090	7.5	0.13	0.989
L4/L5	0.29	21	0.60	480	120	0.098	8.5	0.25	0.985
L5/L6	0.25	29	0.50	490	120	0.090	9.1	0.49	0.969
L6/L7	0.31	64	0.31	820	180	0.068	11.2	0.38	0.955
Tension	0.41	4.5	0.27	1240	120	—	—	0.32	0.997

strain or laxity in the spine that does not immediately recoil once the flexion is terminated. Further, 7 hours of rest were not sufficient to bring about full recovery (>95%) of the lost muscular activity, tension, nor the residual strain. The recovery of the muscular activity consists of initial hyperexcitability on flexion, followed by a complex and slow biexponential increase that predicts that nearly 24 hours are required for full recovery.

It is well established that viscoelastic structures, such as ligaments and discs, exhibit stress–relaxation or creep when subjected to repetitive or prolonged static displacements or loads, respectively.[1,2,6,10,13,16,19,32] In essence, the creep or laxity developed in the viscoelastic structures causes the normal baseline tension within the tissue to decrease. When the baseline tension in the viscoelastic tissues decreases significantly below the excitation threshold of the mechanoreceptors in these tissues,[12,37,38] the initiation of reflexive activity from such receptors to the multifidus muscle[14,15,34] is inhibited, exposing the lumbar spine to motion without the protection of the stiffening muscular forces. Under such circumstances, the potential for large and abnormal displacements of vertebrae relative to each other is possible, and that may be the source of injury. Further, spasms are known to be associated with, or to result from injury and pain.[4,27,29,30] Therefore, although the flexion of the lumbar spine in this investigation was well within the normal range of motion, EMG spasms were recorded from all lumbar levels. The spasms and associated measured residual laxity indicate that some sort of temporary subacute damage may have occurred in any of the viscoelastic tissues. The spasms were most likely initiated from nociceptors that are present in the ligaments and disc.[12,37,38]

Seven hours of rest after the 20 minutes of static flexion allowed the recovery of the tension in the lumbar spine from 32% to only 79% of their initial value, a 47% recovery. Most of the recovery (36% of the total 47%) occurred in the first 10 minutes of rest, and the additional 7 hours resulted in only an additional 11% increase. The residual axial strain in the supraspinal ligament recovered from the 7.15% ± 2.11 calculated at the end of the 20 minutes of flexion to 0.87% ± 0.26 at the end of the 7 hours of rest, confirming that the normal baseline tension in the viscoelastic tissues was not fully restored and that full reflexive muscular activity should not be expected.

The experimental set-up followed recovery for 7 hours. Therefore, it was not possible to determine experimentally if the residual elongation of 0.87% was a plastic deformation. This is an important issue that should be addressed in future work.

The reflexive muscular activity in the L1/L2–L6/L7 lumbar levels recovered from 30, 18, 13, 12, 49, and 38% to 83, 82, 75, 63, 77, and 79%, respectively. Again, the majority of the recovery was recorded within the first 30 minutes of the rest period, and the following 6 hours of rest were characterized with a very slow exponential increase. Full recovery of reflexive muscular activity at the end of 7 hours rest was present only in the multifidus of two to three levels in two of the seven preparations. The model also predicts that nearly 24 hours may be necessary to restore full (> 95%) reflexive muscular activity.

The model fitted to the NIEMG data consists of three terms that are time dependent and a residual. The first term is given in (c) below:

$$A(1 - e^{-\frac{t}{T1}}) \qquad (c)$$

This term describes a relatively fast increasing exponential and most likely accounts for the behavior of the several ligaments and capsules of the lumbar spine. The discs, despite the fact that they are viscoelastic tissues as well, have a different response. Their recovery is described by the term below:

$$B(1 - e^{\frac{-(t-\tau)}{T2}}) \qquad (d)$$

The terms (c) and (d) are different in two significant factors. First, the time constant T2 is much slower than that assigned to T1. Second, a time delay, τ, is associated with the recovery of discs.

It has been well established that repetitive or prolonged static compression/deformation of a disc results in the escape of the fluids from within and that its reabsorption with rest is long and slow.[1,2] Therefore, the static flexion imposed on the lumbar spine may have resulted in significant loss of fluids from the discs during the 20 minutes. Once the rest period had begun, the internal pressure in the disc was decreased because of the absence of some of the fluids. The discs, therefore, had a low contribution to the overall stiffness of the lumbar spine until 2–3 hours later, when sufficient fluids were reabsorbed such that its change in stiffness became noticeable; hence, the time delay τ associated with term (d). Because the recovery of the lost fluids is rather lengthy, on the order of several hours, the time constant T2 associated with (d) is much longer than T1.[1,2] The term (d) describing the recovery of the disc, therefore, exhibits a 2–3 hour delayed impact on the overall recovery, and that recovery is slow and does not arrive to its asymptotic range during the 7-hour recovery period monitored in this investigation.

Whereas the fast and slow exponential terms fully described the recovery of reflexive muscular activity after cyclic loading, a third exponential term was required in this investigation.[7,33] The NIEMG from all lumbar levels consistently exhibited a large initial increase at the beginning of the rest period that was followed by a minor but noticeable and persistent decrease. Overall, the first hour of rest was characterized by an increase and minor decrease in EMG readings before the pattern followed

the exponential terms given in equations (c) and (d). These initial transient phenomena are described by the term given in (b) below:

$$Cte^{\frac{-t}{T3}} \quad \text{(b)}$$

The spasms that persisted in the EMG discharge throughout the 20 minutes of static flexion are probably the manifestation of nociceptor reflex activation caused by temporary subacute damage in the viscoelastic tissues. The initial hour of rest, therefore, was subjected to hyperexcitability of the multifidus muscles attempting to increase the stiffness of the lumbar spine to prevent undue exposure of already strained ligaments and discs to any additional damage. In all lumbar levels, this "neurologic" component (*e.g.*, term (b)), was transitory and completed by the end of the first hour of rest. Because the recovery of the ligaments was relatively rapid, one may tend to assume that some subacute damage was most likely present in these tissues. In all cases, the transient component (b) was over by the time the ligamentous component (c) exhibited most of its recovery (*e.g.*, reached near its asymptotic level). The neurologic component, therefore, seems to account for an additional reflexive loop from nociceptors, with the objective of protecting ligamentous structures, already strained and possibly with some subacute damage, from further exposure.

The tension recorded from the spine during the recovery period did not exhibit the initial transient peak and was fully described by the ligamentous (c) and disc (d) terms of the model as well as the residual.

During anesthetic conditions, respiration is shallow. It may be conceivable that in normal conditions with proper rest, the recovery may have been somewhat faster because the tissues would be better oxygenated. Nevertheless, the pattern of recovery and its components would not be expected to change.

An important issue emerging from this investigation is the fact that some of the EMG spasms were 10–12 times larger in intensity than the initial EMG of the multifidus muscles. Muscular coactivation is an integral response to motion of the spine in various planes,[8,18,21,35,39] as well as motion of the various limb joints,[5,9,31] and such coactivation is for purposes of insuring joint stability.[11,20] The normal coactivation level observed in the spine and other limb joints is in the order of 5–15% of the maximal voluntary contraction (MVC) level. The fact that some of the observed spasms were 10–12 times larger than the initial reflexive EMG asserts that reflexive activation of antagonist muscles by mechanoreceptors in ligament and other viscoelastic structures is a major component of coactivation. It also could be asserted from the spasms that the initial activation of the multifidus was at the 10% (of maximal) level and was the coactivation one can expect in normal activities.

■ Conclusion

After static flexion, the recovery of multifidus activity with rest is characterized by an initial period of hyperexcitability, followed by a biexponential increase. The biexponential increase consists of the rapid recovery of ligamentous tissues and of a slow and delayed recovery associated with fluid reabsorption in discs. The full recovery of reflexive multifidus activity and viscoelastic tension with rest after a short 20 minutes of static flexion may require more than 24 hours.

Finally, it could be inferred that static flexion of the lumbar spine in occupational and sports activities is an extremely imposing function on its viscoelastic tissues because it is associated with severe tension–relaxation, residual strain, decrease in muscular stabilizing forces, and spasms. These implications of static lumbar flexion, even within the physiologic range and for relatively short periods, may expose the spine to potential disorders and require extremely long periods of rest before normal functions are reestablished.

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