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Neuromuscular Response to Cyclic Lumbar Twisting

Li Li, Nirav Patel, Deborah Solomonow, Peter Le, Heather Hoops, David Gerhardt, Kyle Johnson, Bing He Zhou, Yun Lu and Moshe Solomonow

Human Factors: The Journal of the Human Factors and Ergonomics Society, October , 2007; 49 (5): 820-829.

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Neuromuscular Response to Cyclic Loading of the Anterior Cruciate Ligament

Paola Sbriccoli, Moshe Solomonow, Bing-He Zhou, Yun Lu and Robert Sellards

Am J Sports Med, April , 2005; 33 (4): 543-551.

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HIGHLIGHTED TOPIC | *Neural Control of Movement*

Longer static flexion duration elicits a neuromuscular disorder in the lumbar spine

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LaBry, Rebecca, Paola Sbriccoli, Bing-He Zhou, and Moshe Solomonow. Longer static flexion duration elicits a neuromuscular disorder in the lumbar spine. *J Appl Physiol* 96: 2005–2015, 2004. First published January 23, 2004; 10.1152/japplphysiol.01190.2003.—The objective of this study was to assess the impact of two sequential long, static, anterior lumbar flexions on the development of a neuromuscular disorder and to compare it with previously obtained data from a series of short static flexion periods of the same cumulative time (Sbriccoli P, Solomonow M, Zhou BH, Baratta RV, Lu Y, Zhu MP, and Burger EL, *Muscle Nerve* 29: 300–308, 2004). Static flexions with loads of 20, 40, and 60 N were applied to the lumbar spine over two 30-min periods with a 10-min rest in between. The reflex EMG activity from the multifidus muscles and supraspinous ligament displacement (creep) was recorded during the flexion periods. Creep and EMG were also monitored over 7 h of rest following the work-rest-work cycle. It was found that the creep that developed in the first 30-min flexion period did not recover completely during the following 10 min of rest, giving rise to a large cumulative creep at the end of the work-rest-work session. Spasms were frequently seen within the EMG during the static flexion. Initial and delayed hyperexcitabilities were observed in all of the preparations at any of the three loads explored during the 7-h rest period. ANOVA revealed a significant effect of time ($P < 0.0001$) on the postloading data. Larger loads elicited larger magnitudes of the initial and delayed hyperexcitabilities, yet were not statistically different. It was concluded that the 3:1 work-to-rest duration ratio resulted in a neuromuscular disorder, regardless of the load magnitude. The conclusions are reinforced in view of the results from a previous study using 60 min of flexion overall but at 1:1 work-to-rest ratio in which only the highest load elicited a delayed hyperexcitability (Sbriccoli et al., *Muscle Nerve* 29: 300–308, 2004). An optimal dose-to-duration ratio needs to be established to limit, attenuate, or prevent the adverse effects of static load on the lumbar spine while considering the loading duration as a major risk factor.

electromyography; muscle; lumbar; spine; disorder

CUMULATIVE TRAUMA DISORDER (CTD) is subjectively characterized by joint pain, limited range of motion, and weakness (inability to generate forces or sustain loads within the physiological range). In the spine, objective diagnostic procedures fail to identify vertebral fractures, herniated disk, facet joint impingement, or stenosis within the canal, and so forth. The epidemiological literature demonstrates that workers exposed

to static, cyclic, and vibratory occupational activities for extended periods of time develop CTD (8, 19, 24). The epidemiology goes on to demonstrate additional correlations to the duration and magnitude of loads developed in such activities and the number of repetitions of the activity within a given day. Physiological and biomechanical experimental validation of the disorder and its dependence of the various risk factors, as well as the tissues involved, is lacking.

Overall, it seems that a dose-duration formula may exist that may predict the relationships between CTD and the magnitude of loads developed within the joint tissues, the duration over which the loads were applied, and the number of repetitions that such load over time was executed (6). Intuitively, a rest period between sequential episodes of load sustained over time may also play a prominent role in the development or prevention of CTD. Similarly, the overall period, in months or years, that a worker was exposed to such activity may also be incorporated as a major component of any dose-duration formula (15, 16).

Our laboratory's initial research related to this issue developed a feline model that could be used to gain new insights on the biomechanics and physiology of CTD in the lumbar spine (2, 21, 28, 30, 32). So far, it was shown that passive static or cyclic flexion of the lumbar spine subjected to 10- to 20-min load resulted in the development of substantial creep in the viscoelastic tissues (ligaments, disks, facet capsule, dorsolumbar fascia, etc.) during the flexion period and only partial recovery of the creep over 7 h of following rest. The electromyographic (EMG) manifestations of the flexion consisted of a five-component neuromuscular disorder (26, 27). Exponentially decreasing reflexive EMG was present during the flexion period with randomly appearing spasms. During the following 7-h rest, short-loading tests of 2 s revealed initial (during the 1st h) and delayed (3–7 h into the rest) long-term hyperexcitabilities. A slow exponential recovery of the EMG to its normal level was also present throughout the 7 h of rest. Pathological analysis of the lumbar supraspinous ligaments revealed that an acute inflammation gradually developed in the tissue, peaking in the 6–7 h of rest, as evidenced by as much as 100 times higher neutrophil density than in controls (26). The data demonstrated that the delayed hyperexcitability and the increase in neutrophil density were developing in parallel, suggesting that this hyperexcitability is the manifestation of the

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acute inflammation. It is conceivable that the ongoing presence of acute inflammation in the spinal viscoelastic tissues will eventually become a chronic inflammation and result in the tissues' degeneration as well as deterioration of its mechanical properties, displaying the CTD signs of limited range of motion, weakness, and pain (12, 15, 16).

Additional insights regarding CTD and its risk factors demonstrated that larger loads and more repetitions are associated with a significant increase in initial and delayed hyperexcitability and, therefore, acute inflammation (21, 22). Low loads and fewer repetitions do not elicit the delayed hyperexcitability and/or inflammatory response when a schedule of 10-min work and 10-min rest was repeated three and six times but was present for nine times (22).

It is of interest to gain insight on the role of various durations of load application as a risk factor for the development of delayed hyperexcitability and/or acute inflammation and, therefore, CTD in the long term. Because data relating the development of the neuromuscular disorder to a schedule of 10-min work and 10-min rest repeated six times (for a cumulative work time of 60 min) are available for three loads spanning the physiological range (20, 40, and 60 N) (21), a different schedule could be explored for comparison. A schedule of two 30-min work spaced by a 10-min rest, for example, will also allow a cumulative work time of 60 min, yet with a longer continuous exposure to the load and less rest.

It is hypothesized that passive spinal flexion with loads within the physiological range applied for two periods of 30 min with a 10-min rest interval will elicit a neuromuscular disorder with a prominent delayed hyperexcitability and that larger load magnitudes will further contribute to the severity of the hyperexcitability. The additional insight gained from such information may have sufficient impact on defining the array of risk factors of CTD and the design of work schedules that limit, attenuate, or prevent the disorder.

METHODS

Preparation. Twenty adult cats, 1–2 yr old, with average weight of 4.51 kg, were used in this study. Cats were anesthetized with 60 mg/kg chloralose, according to a protocol approved by the Institutional Animal Care and Use Committee. The skin overlying the lumbar spine was dissected to expose the lumbar fascia, and an S-shaped stainless steel hook made of 1.5-mm-diameter rod was applied around the supraspinous ligament between L₄ and L₅. The preparation was then positioned in a rigid stainless steel frame and fixed for subsequent EMG electrode insertion. Preparations were divided into three experimental groups, each subjected to a different load: 20 N ($n = 6$) for the first group, 40 N ($n = 7$) for the second group, and 60 N ($n = 7$) for the third group.

Instrumentation. The lumbar spine was isolated by means of two external fixators, which were applied to the L₁ and L₇ posterior process, respectively. The external fixation was intended to limit the elicited flexion to the lumbar spine and to prevent interaction of thoracic and sacral and/or pelvic structures but not to prevent any motion.

Three pairs of fine stainless steel wire EMG electrodes (interelectrode distance: 3–4 mm) were inserted in the right L₃–L₄, L₄–L₅, and L₅–L₆ multifidus muscles 8 mm laterally from the posterior spinal processes. A ground electrode was inserted into the gluteus muscle. Each electrode pair constituted the input to a differential EMG amplifier with a 110-dB common mode rejection ratio, a gain of up to 200,000, and a band-pass filter in the range of 6–500 Hz. The EMG was recorded with a sampling rate of 1,000 Hz, and it was continu-

ously monitored on an oscilloscope. The S-shaped stainless steel hook inserted around the L₄–L₅ supraspinous ligament was connected to the crosshead of the Bionix 858 Material Testing System (MTS, Minneapolis, MN), in which a load cell was located. The load was applied through the MTS actuator with a computer-controlled loading system. The vertical displacement of the actuator was also monitored continuously. The load cell and displacement outputs of the Bionix 858 MTS were sampled into the computer along with the EMG signals.

Protocol. The three experimental groups were subjected to the protocol described below. A pretension of 1 N was applied to the supraspinous ligament to standardize the initial conditions in all of the different preparations (4). For each experimental group, a different constant load (20, 40, or 60 N) was applied to the lumbar spine via the S-shaped stainless steel hook. These different loads were chosen because they covered the range between minimum (just above the reflex excitation threshold load of the ligament) and maximum physiological strain, as previously described (2, 30). The tension level was maintained constant during a 30-min loading period, followed by 10-min rest and by a second 30-min loading period. The EMG signal, the vertical displacement, and the load were recorded continuously during the loading periods. Nine 8-s loading tests were performed during the following 7 h of recovery. Each group was loaded during the recovery with the same load used in the two 30-s tests (see Fig. 1). This was obtained by a linear increase in tension over 6 s followed by 2 s of constant load. The linear increase in load over 6 s was used to avoid possible damage to the ligaments due to a sudden or fast stretch (17).

A 6-s ramp to the respective load of each of the three experimental groups was also applied in the initial loading of the 30-min working periods.

The same protocol was used for the three different loads (20, 40, and 60 N), and each preparation was subjected to only one load. The EMG, load, and supraspinous ligament displacement data were then stored in the computer for subsequent analysis.

Data analysis. The analysis of the EMG, vertical displacement, and the static load applied to the supraspinous ligament was performed over 1.5-s epochs. During the two 30-min constant loads, the analysis was performed at the very beginning of the loading period and then every 20 s for each 30-min static load. During the recovery phase, the analysis was performed over the 2-s constant-load phase following the 6-s ramp. To be confident that the load was fully applied, the first 0.5 s of the constant-load phase (2-s length) was discarded, and the analysis was performed over the following 1.5 s. Each EMG sample was integrated over the 1.5-s epoch and normalized with respect to the integrated EMG computed for the first window of the first 30-min constant load to obtain the normalized integrated EMG (NIEMG). For each experimental group, all of the corresponding NIEMG data were pooled together, and the mean and SD values were computed and plotted on NIEMG vs. time for each of the muscles of the three lumbar levels investigated. The displacement data were normalized to the displacement recorded at the beginning of the first 30-min loading period. Then the corresponding normalized displacement data of each of the experimental groups (20, 40, and 60 N) were pooled together as means (\pm SD) and plotted as normalized displacement vs. time.

Model. The model considered is based on our laboratory's previous work in which continuous 20-min static load was followed by a 7-h recovery period (27, 29, 31). To convert the equations to describe a series of work periods spaced by rest periods, two new time components are defined. T_w is the time period over which work (or load) was performed (or applied) by (to) the spine, which was 30 min in this study. T_R is the period of rest between the two work periods (T_w), 10 min in this study. The equation describing the NIEMG behavior during each of the work periods is rewritten as

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

where NIEMG(t) is the NIEMG as a function of time t ; NIEMG₀ is the steady-state amplitude of the NIEMG; A_n is the amplitude of the

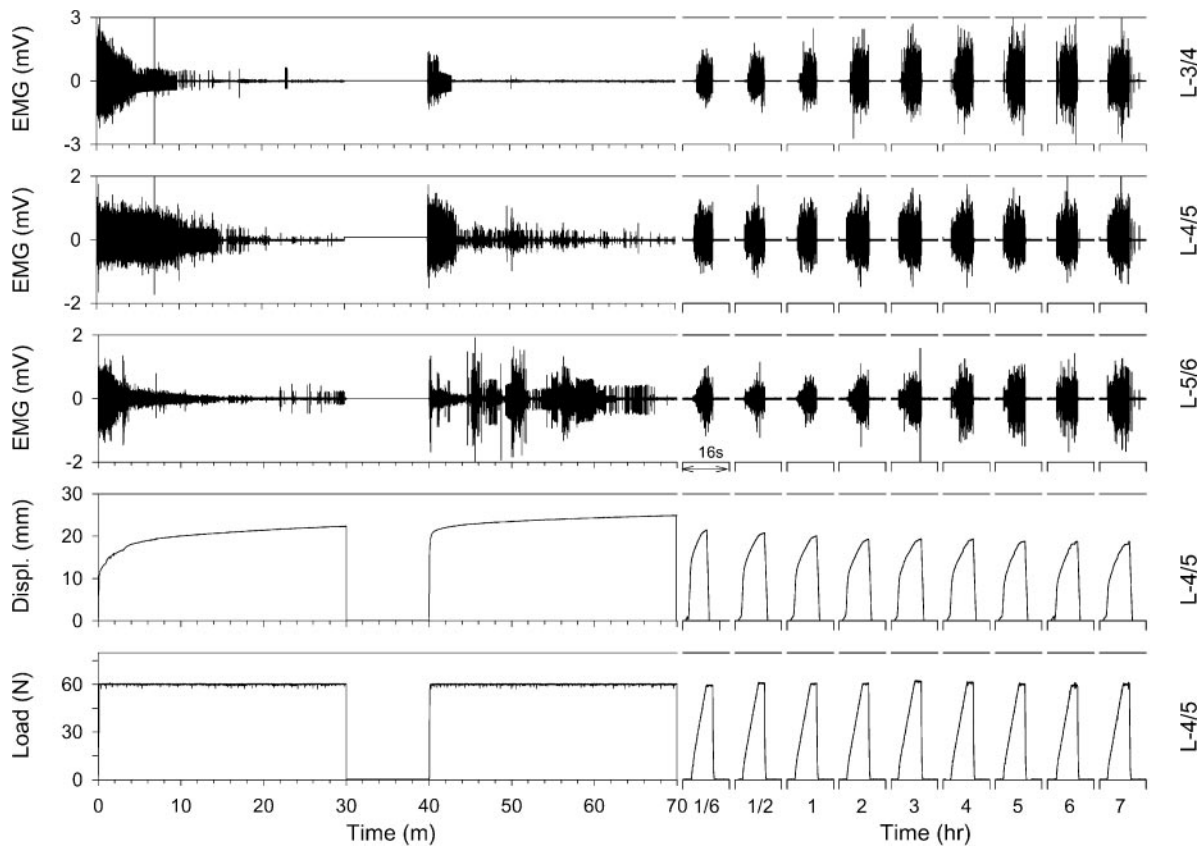


Fig. 1. Typical recording of electromyography (EMG) from the L₃-L₄, L₄-L₅, and L₅-L₆ multifidus (top 3 rows) and lumbar displacement (Displ.) and static load (bottom 2 rows) recorded from 1 preparation subjected to a load of 60 N.

exponential component of the NIEMG; and T_{n1} is the time constant of the exponential component.

It was assumed that A and $NIEMG_0$ are not constant throughout the work-rest session, i.e., A and $NIEMG_0$ are changing from one work period to the next. It was also assumed that T_1 might not be the same for the two work periods.

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

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

where B_n is the amplitude of the exponential component of the NIEMG during recovery, and T_{n2} is the time constant of the exponential.

The equation describing the development of displacement (Disp) (and indirect creep in the viscoelastic tissues) during the two work periods spaced by a rest period is given by

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

where D_{0n} is the elastic component of amplitude; D_{Ln} is the viscoelastic component amplitude; and T_{n5} is the time constant of the creep during flexion.

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

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

where R_n is the residual creep at the end of each rest session, and T_{n6} is the time constant governing the recovery of creep in each rest session.

The long-term 7-h recovery after the work-rest-work session was modeled by the original equation for long-term recovery (2, 27).

Once the means \pm SD of the experimental data were calculated, attempts were made to generate the best-fit models described above by using the Marquardt-Levenberg nonlinear regression algorithm. In some cases, the algorithm failed to converge satisfactorily; in these cases, initial or final values were determined by sequential recursive iteration, optimizing for regression coefficient.

To test for the effect of the three protocols adopted (20, 40, and 60 N) on the NIEMG and displacement data at the three lumbar levels explored (L₃-L₄, L₄-L₅, and L₅-L₆), a Fisher's post hoc test was adopted. Furthermore, a two-way ANOVA was performed to assess the effect of time postloading and load magnitude on the recovery of the NIEMG and the displacement. Significance was set at 0.05 for all statistical tests.

RESULTS

A typical example of EMG, load, and displacement from a preparation subjected to 60 N is shown in Fig. 1. In the first 30 min of loading, the EMG is progressively decreasing over time for all the three lumbar levels, with the decrease being more evident during the second 30-min loading period. Note the presence of random spasms during the two loading periods and during the 7 h of recovery.

The mean (\pm SD) NIEMG and displacement data collected for 20, 40, and 60 N are shown in Figs. 2, 3, and 4, respectively.

In the group subjected to 20 N (Fig. 2), the mean displacement at the beginning of the first 30-min load was 5.96 mm and reached a final value of 11.809 mm. The resulting creep was 98.13%. The creep partially recovered during the 10-min rest to a mean value of 10.297 mm. At the end of the two working periods, the mean displacement was 12.727 mm, corresponding to a creep of 113.54%. During the 7 h of recovery, the creep decreased continuously, and the residual creep at the end of the recovery phase was 34.96%.

The mean displacement developed in the preparations subjected to 40 N (Fig. 3) was 10.765 mm at the beginning of the first working period, reaching a mean value of 17.929 mm (mean creep: 66.54%) at the end of the first 30-min load. During the 10-min rest, the creep recovered to a mean value of 41.97%. The displacement further increased during the second working period, up to a mean final value of 19.052 mm, corresponding to a mean creep of 76.98%. During the 7 h of recovery, the displacement decreased to a mean final value of 11.642 mm, resulting in a mean residual creep of 8.14%.

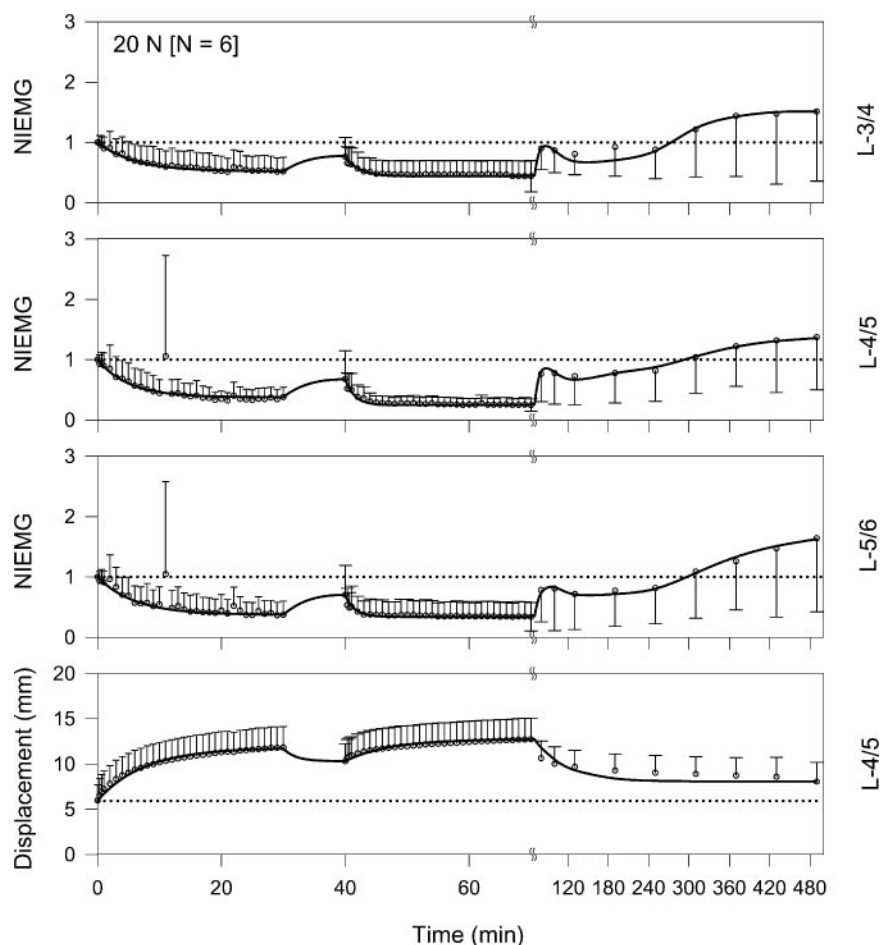
For the group subjected to 60 N (Fig. 4), the mean initial displacement was 13.014 mm, and the mean value reached during the first 30-min load was 20.517 mm, which corresponds to a mean creep of 57.65%. The 10-min rest allowed a partial recovery of creep to 40.37%. At the end of the two working sessions, the mean displacement was 18.269 mm, corresponding to a mean creep of 40.7%. A gradual decrease of

the displacement to a mean final value of 16.158 mm (residual creep: 24.1%) was observed during the 7 h of recovery.

The mean NIEMG decreased during the first working 30-min period in the preparations subjected to 20 N from 1.0 to 0.518 (48.2% decrease), 0.378 (62.2% decrease), and 0.372 (62.8% decrease) at L₃-L₄, L₄-L₅, and L₅-L₆, respectively. A partial recovery of the NIEMG was observed during the 10 min of rest to mean values of 0.763, 0.677, and 0.696 at the three lumbar levels inspected. During the following 30-min load, the NIEMG further decreased, reaching mean values of 0.439 (L₃-L₄), 0.246 (L₄-L₅), and 0.339 (L₅-L₆). During the first 10 min of the 7 h of recovery, the NIEMG showed a sharp increase to mean values of 0.898 at L₃-L₄ and 0.781 for both L₄-L₅ and L₅-L₆. This was followed by a slight decrease in the NIEMG during the following 2 h of recovery. Afterward, the NIEMG gradually increased, reaching mean values of 1.509 at L₃-L₄, 1.371 at L₄-L₅, and 1.638 at L₅-L₆ at the end of the 7 h of rest. It is important to note that these final values are higher than the initial NIEMG of 1.0 at all of the lumbar levels considered and that the 1.0 value was reached, on average, after 4 h of rest.

For the preparations subjected to 40 N, the NIEMG showed an initial decrease during the first working session, reaching mean values of 0.359, 0.309, and 0.317 at L₃-L₄, L₄-L₅, and L₅-L₆, respectively. A partial recovery of the NIEMG was observed as well during the 10 min of rest between the two

Fig. 2. Mean (\pm SD) normalized integrated EMG (NIEMG) of the L₃-L₄, L₄-L₅, and L₅-L₆ multifidus as well as the mean displacement during the load-rest-load (30:10:30 min) session and the 7-h recovery are shown for the load of 20 N. Solid lines through the data points represent the models developed to describe NIEMG and displacement during work-rest and long-term recovery. During the 30 min of loading, only 35 data points are presented for figure clarity.



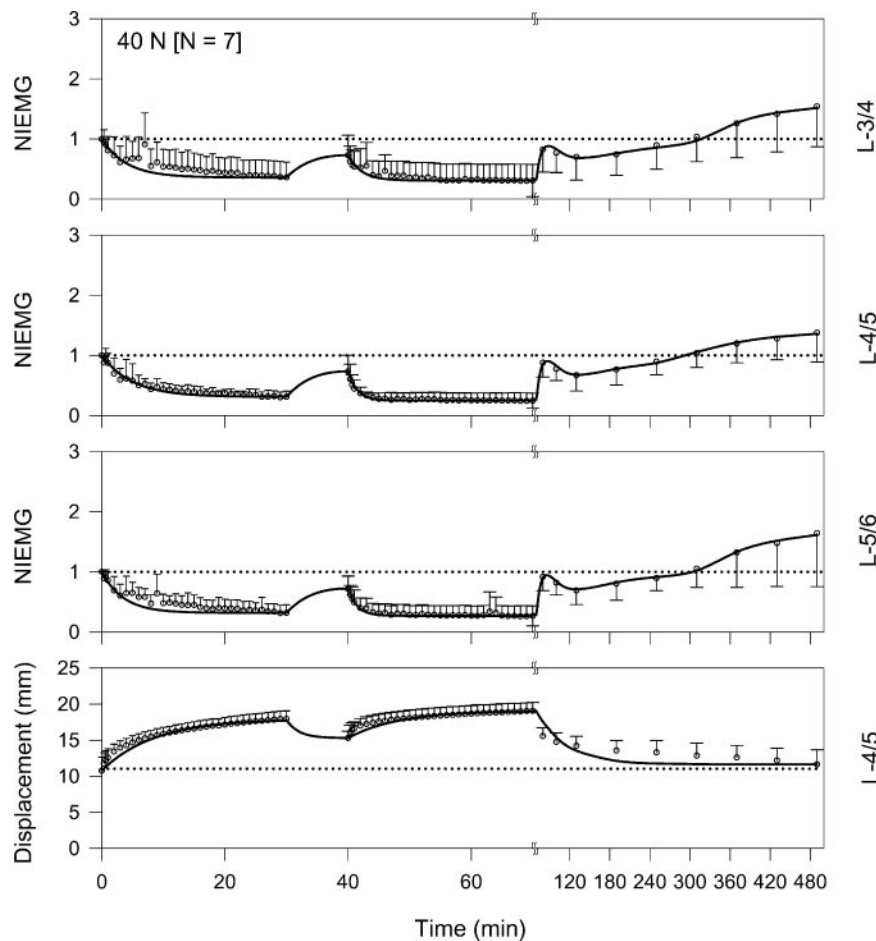


Fig. 3. Mean (\pm SD) NIEMG of the L₃-L₄, L₄-L₅, and L₅-L₆ multifidus as well as the mean displacement during the load-rest-load (30:10:30 min) session and the 7-h recovery are shown for the load of 40 N. Solid lines through the data points represent the models developed to describe NIEMG and displacement during work-rest and long-term recovery. During the 30 min of loading, only 35 data points are presented for figure clarity.

working sessions. The second working session resulted in a further decrease in the NIEMG, and the mean values at the end of the two working periods were 0.307 (69.3%), 0.246 (75.4%), and 0.267 (73.3%) for the three lumbar levels. The 7 h of recovery consisted of an initial NIEMG peak to values of 0.824, 0.876, and 0.917 in the 1st h for the three lumbar levels considered. The NIEMG peak was followed by a gradual decrease during the following 2 h of recovery. The NIEMG gradually then increased, exceeding 1.0 after the 4th h for all of the three lumbar levels. The mean final NIEMG values at the end of the 7-h recovery phase were 1.542 at L₃-L₄, 1.375 at L₄-L₅, and 1.643 at L₅-L₆.

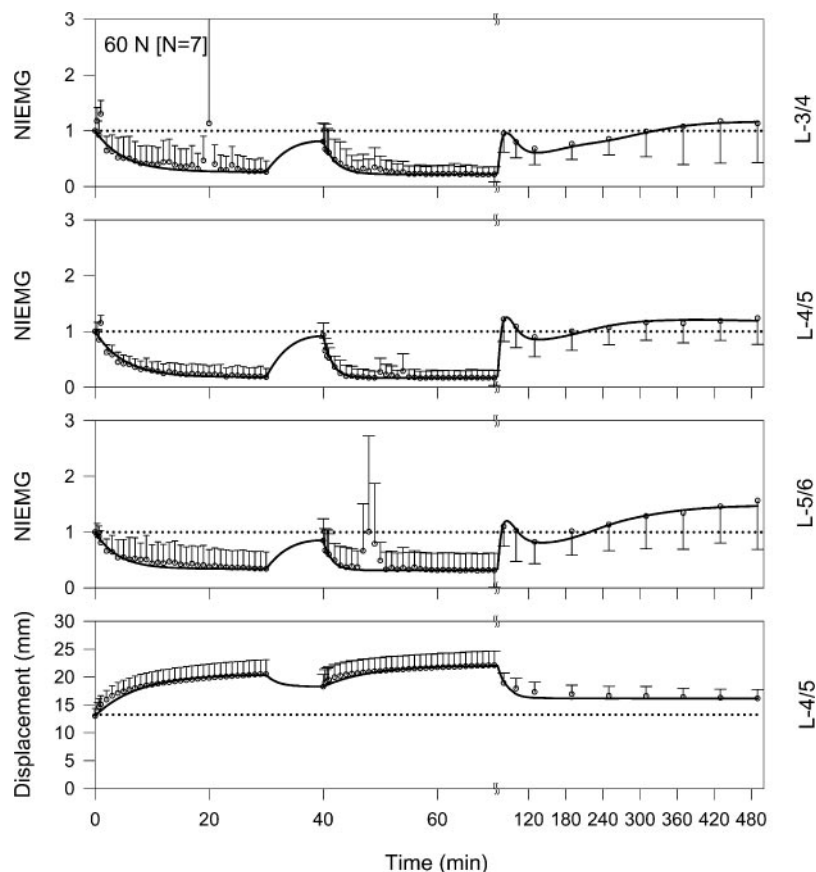
For the preparations subjected to 60-N load, the multifidus NIEMG showed a marked decrease during the first working session to values of 0.259 (74.1% decrease), 0.178 (82.2% decrease), and 0.338 (66.2% decrease) at L₃-L₄, L₄-L₅, and L₅-L₆, respectively. During the 10 min of rest between the two working sessions, the NIEMG recovered to 0.807, 0.935, and 0.845 at the three lumbar levels inspected. The following 30-min working session resulted in a decrease of the mean NIEMG for the three lumbar levels. The NIEMG decrease was steeper than that observed during the first working session and in the other two experimental groups (20- and 40-N load) as well, and the mean NIEMG value obtained at the end of the two working sessions was 0.216 (L₃-L₄), 0.165 (L₄-L₅), and 0.313 (L₅-L₆), corresponding to a mean NIEMG decrease of 78.4, 83.5, and 68.7%, respectively. As it was observed in the

other two groups, an initial NIEMG peak characterized the 1st h of recovery, with mean NIEMG values ranging from 0.951 (L₃-L₄) to 1.217 (L₄-L₅) and 1.102 (L₅-L₆), and by a subsequent NIEMG decrease. Thereafter, a gradual increase in the NIEMG was observed in the three lumbar levels investigated, and the 1.0 preload value was reached after the 2nd h of recovery at L₄-L₅ (1.001) and L₅-L₆ (1.016). The mean NIEMG values observed at the end of the 7 h of recovery were 1.131, 1.236, and 1.566 at L₃-L₄, L₄-L₅, and L₅-L₆, respectively.

The best-fit model constants fitted to the NIEMG, and displacement data were superimposed on the mean (\pm SD) of the experimental data presented in Figs. 2-4. The model constants are reported in Tables 1, 2, and 3 for the 20-, 40-, and 60-N preparations, respectively.

For the group subjected to 20 N, A_n , T_{n1} , and $NIEMG_{0n}$ changed between the two working periods (Table 1). The time constant T_{n1} showed the most relevant changes, decreasing (in minutes) between the two working periods from 6 to 2.2 for the L₃-L₄, from 5 to 1.5 for the L₄-L₅, and from 6 to 1.5 for the L₅-L₆. The $NIEMG_{0n}$ in the two working periods exhibited minor changes, slightly increasing and decreasing, for the three lumbar levels (from 0.974 to 0.872 in L₃-L₄, 0.758 to 0.938 in L₄-L₅, and 0.736 to 0.872 in L₅-L₆), suggesting the absence of trend. $NIEMG_{0n}$, therefore, could be considered as unchanged. The constant A_n decreased in all of the three lumbar levels studied, from 0.479 to 0.321 for the L₃-L₄, from 0.620 to 0.430

Fig. 4. Mean (\pm SD) NIEMG of the L₃-L₄, L₄-L₅, and L₅-L₆ multifidus as well as the mean displacement during the load-rest-load (30:10:30 min) session and the 7-h recovery are shown for the load of 60 N. Solid lines through the data points represent the models developed to describe NIEMG and displacement during work-rest and long-term recovery. During the 30 min of loading, only 35 data points are presented for figure clarity.



for the L₄-L₅, and from 0.624 to 0.357 for L₅-L₆. During the 10 min of rest between the two working sessions, B_n was fairly constant among the three lumbar levels, with values ranging from 0.07 to 0.09. The time constant T_{n2} was 10 min for all of the three lumbar levels considered. According to the model developed for the displacement data during the two working sessions, the time constant T_{n5} was nearly constant at 7 min. D_{0n} increased from 5.960 to 10.297 mm, whereas D_{Ln} decreased from 4.307 to 1.848 mm.

Similar changes in the model parameters were found for the NIEMG and displacement models in the groups subjected to 40- and 60-N load during the working periods (see also Tables 2 and 3).

During the 7 h of recovery, the models developed for the NIEMG data showed some variations between groups. The time constant T_2 , representative of the initial hyperexcitability, was substantially modified among the three different groups at any of the lumbar levels, being lower in the 60-N group (mean T_2 : 12.66 min) compared with the groups subjected to 40 N (mean T_2 : 14 min) and 20 N (mean T_2 : 16 min). The time constant T_3 , governing the steady-state recovery phase, showed a progressive marked decrease from the mean value of 146 min obtained in the 20-N group to the values of 100 and 76.6 min observed for the 40- and 60-N groups, respectively. The time constants T_4 and T_d , both representative of the delayed hyperexcitability, were lower in the preparations subjected to 60-N load (T_4 : 237 min; T_d : 225 min), suggesting that an earlier and faster development of the delayed hyperexcitability was occurring in this group. The R^2 found for the models developed for

NIEMG and displacement were in good agreement with the experimental data, with values above 0.9. Some exceptions were present and were ascribed either to the presence of spasms interrupting the smooth changes in the EMG discharge or to the high standard deviation found (L₅-L₆, 60-N preparations).

Although some differences were evident in the mean data of the three protocols adopted (20, 40, and 60 N), these changes were not supported by the statistical analysis. The post hoc Fisher's test failed to show any significant difference between different loads at any of the three lumbar levels; according to the ANOVA analysis, only a time effect ($P < 0.0001$) was found among the three different groups, indicating that all of the considered parameters (NIEMG L₃-L₄, L₄-L₅, and L₅-L₆ and displacement) were changing as time progressed. No effect of load and no interaction effects were indeed found.

DISCUSSION

The most relevant finding of the present study was that a neuromuscular disorder was elicited in feline preparations after a static load was applied to the lumbar spine for as long as two 30-min periods spaced by 10-min rest. The delayed hyperexcitability component of neuromuscular disorder was present after 7 h of recovery in all of the preparations, regardless of the load magnitude (20, 40, or 60 N). Interestingly, despite the observed changes among the three different experimental groups, they were not statistically significant, indicating that the load magnitude was not the major determinant in promot-

Table 1. *Model parameters for 20-N load*

NIEMG During Work Period $NIEMG(t) = A_n e^{-[t - n(T_W + T_R)]/T_{n1}} + NIEMG_{0n}$						
20 N	<i>n</i>	<i>A_n</i>	<i>T_{n1}</i> , min	<i>NIEMG_{0n}</i>	<i>r</i> ²	
L ₃ -L ₄	0	0.479	6	0.521	0.974	
	1	0.321	2.2	0.442	0.872	
L ₄ -L ₅	0	0.620	5	0.380	0.758	
	1	0.430	1.5	0.247	0.938	
L ₅ -L ₆	0	0.624	6	0.376	0.736	
	1	0.357	1.5	0.339	0.872	

NIEMG During Rest Period $NIEMG(t) = \{t - [(n + 1)T_W + nT_R]\}B_n e^{-[t - (n+1)T_W + nT_R]/T_{n2}} + NIEMG_{0n}$				
20 N	<i>n</i>	<i>NIEMG_{0n}</i>	<i>B_n</i>	<i>T_{n2}</i>
L ₃ -L ₄	0	0.519	0.07	10
L ₄ -L ₅	0	0.380	0.08	10
L ₅ -L ₆	0	0.372	0.09	10

NIEMG During 7-h Recovery $NIEMG(t) = E(1 - e^{-t/T_3}) + tB e^{-t/T_2} + C(t - T_d)e^{-[t - T_d]/T_4} + NIEMG_0$								
Level	<i>E</i>	<i>T₃</i> , min	<i>B</i>	<i>T₂</i> , min	<i>C</i>	<i>T₄</i> , min	<i>T_d</i> , min	<i>r</i> ²
L ₃ -L ₄	0.561	190	0.085	15	0.008	200	240	0.942
L ₄ -L ₅	0.754	100	0.1	14	0.0035	300	270	0.991
L ₅ -L ₆	0.661	150	0.06	19	0.0055	370	270	0.990

Displacement During Work Period $Disp(t) = D_{0n} + D_{Ln}\{1 - e^{-[t - n(T_W + T_R)]/T_{n5}}\}$				
<i>n</i>	<i>D_{0n}</i> , mm	<i>D_{Ln}</i> , mm	<i>T_{n5}</i> , min	<i>r</i> ²
0	5.960	4.307	7.5	0.981
1	10.297	1.848	7	0.920

Displacement During Rest Period $Disp(t) = D_{0n} + R_n + (D_{Ln} - R_n)e^{-[t - (n+1)T_W + nT_R]/T_{n6}}$				
<i>n</i>	<i>D_{0n}</i> , mm	<i>D_{Ln}</i> , mm	<i>R_n</i> , mm	<i>T_{n6}</i> , min
0	5.960	4.307	4.337	2

Displacement During 7-h Recovery $Disp(t) = D_0 + R + (D_L - R)e^{-t/T_6}$				
<i>D₀</i> , mm	<i>D_L</i> , mm	<i>R</i> , mm	<i>T₆</i> , min	<i>r</i> ²
5.960	6.797	2.084	46	0.705

See METHODS for definition of terms.

ing the disorder. In view of the previous work on this subject, the longer work period seems to be the major factor in eliciting the long-term component of the disorder. Most importantly, the 3:1 work-to-rest duration ratio turned out to be unfavorable and crucial in promoting the development of a low-back disorder.

As previously observed (26, 27), the neuromuscular disorder provoked manifested itself as a marked decrease in the EMG and spasms during the working periods. The rest period (7 h) was characterized by the presence of an initial and a delayed hyperexcitability, and, at the end of the 7 h of recovery, the NIEMG in all cases were higher than the initial values (e.g., 1.0), suggesting that a severe neuromuscular disorder was induced due to acute inflammation in the viscoelastic tissues (26, 27, 31). Spasms were frequently and randomly observed within the EMG signals, confirming what was already found before (Refs. 18, 29, and 33, among others) and indicating that tissue microdamage existed (10, 18, 33, 34).

The impact of various aspects of static load on the lumbar spine as a main determinant in neuromuscular disorder development has been previously shown by experimental studies (10, 26, 27, 29, 31, 33), as well as epidemiological surveys (3, 8, 13, 15, 16). It was shown experimentally that high-load magnitude and a high number of repetitions are risk factors for the development of a neuromuscular disorder (21, 22).

As shown by the present data, a neuromuscular disorder was provoked as a consequence of the prolonged static load applied to the lumbar spine at any of the three loads (20, 40, and 60 N). The magnitude of the changes in the neuromuscular disorder induced has shown a minor change among different loads, indicating a trend toward a more severe injury at the higher loads (40 and 60 N). During the two 30-min periods of loading, the NIEMG decreased, on average, by 65.86, 72.66, and 78.86% of the preload values for the loads of 20, 40, and 60 N, respectively, confirming what was previously observed (for a

Table 2. *Model parameters for 40-N load*

NIEMG During Work Period $NIEMG(t) = A_n e^{-[t - n(T_W + T_R)]/T_{n1}} + NIEMG_{0n}$						
40 N	<i>n</i>	<i>A_n</i>	<i>T_{n1}</i> , min	<i>NIEMG_{0n}</i>	<i>r</i> ²	
L ₃ -L ₄	0	0.641	4	0.359	0.581	
	1	0.419	2.2	0.311	0.814	
L ₄ -L ₅	0	0.689	5	0.311	0.958	
	1	0.484	2.2	0.247	0.971	
L ₅ -L ₆	0	0.683	5	0.317	0.715	
	1	0.445	1.5	0.268	0.945	

NIEMG During Rest Period $NIEMG(t) = \{t - [(n + 1)T_W + nT_R]\}B_n e^{-[t - (n+1)T_W + nT_R]/T_{n2}} + NIEMG_{0n}$				
40 N	<i>n</i>	<i>NIEMG_{0n}</i>	<i>B_n</i>	<i>T_{n2}</i>
L ₃ -L ₄	0	0.361	0.1	10
L ₄ -L ₅	0	0.311	0.115	10
L ₅ -L ₆	0	0.317	0.11	10

NIEMG During 7-h Recovery $NIEMG(t) = E(1 - e^{-t/T_3}) + tB e^{-t/T_2} + C(t - T_d)e^{-[t - T_d]/T_4} + NIEMG_0$								
Level	<i>E</i>	<i>T₃</i> , min	<i>B</i>	<i>T₂</i> , min	<i>C</i>	<i>T₄</i> , min	<i>T_d</i> , min	<i>r</i> ²
L ₃ -L ₄	0.693	115	0.09	15	0.006	250	300	0.991
L ₄ -L ₅	0.754	100	0.11	14	0.0035	300	270	0.994
L ₅ -L ₆	0.733	85	0.12	13	0.0065	275	300	0.996

Displacement During Work Period $Disp(t) = D_{0n} + D_{Ln}\{1 - e^{-[t - n(T_W + T_R)]/T_{n5}}\}$				
<i>n</i>	<i>D_{0n}</i> , mm	<i>D_{Ln}</i> , mm	<i>T_{n5}</i> , min	<i>r</i> ²
0	10.765	4.955	8.5	0.943
1	15.284	2.688	8	0.841

Displacement During Rest Period $Disp(t) = D_{0n} + R_n + (D_{Ln} - R_n)e^{-[t - (n+1)T_W + nT_R]/T_{n6}}$				
<i>n</i>	<i>D_{0n}</i> , mm	<i>D_{Ln}</i> , mm	<i>R_n</i> , mm	<i>T_{n6}</i> , min
0	10.765	4.955	4.519	2

Displacement During 7-h Recovery $Disp(t) = D_0 + R + (D_L - R)e^{-t/T_6}$				
<i>D₀</i> , mm	<i>D_L</i> , mm	<i>R</i> , mm	<i>T₆</i> , min	<i>r</i> ²
10.765	8.287	0.877	44	0.719

See METHODS for definition of terms.

Table 3. Model parameters for 60-N load

NIEMG During Work Period $NIEMG(t) = A_n e^{-(t-n(T_W+T_R))/T_{n1}} + NIEMG_{0n}$					
60 N	<i>n</i>	<i>A_n</i>	<i>T_{n1}</i> , min	<i>NIEMG_{0n}</i>	<i>r</i> ²
L ₃ -L ₄	0	0.741	4	0.259	0.511
	1	0.585	2.2	0.222	0.935
L ₄ -L ₅	0	0.822	3.7	0.178	0.919
	1	0.770	1.2	0.165	0.948
L ₅ -L ₆	0	0.662	3.5	0.338	0.884
	1	0.531	1.5	0.314	0.175

NIEMG During Rest Period $NIEMG(t) = \{t - [(n+1)T_W + nT_R]\} B_n e^{-(t-[(n+1)T_W+nT_R])/T_{n2}} + NIEMG_{0n}$				
60 N	<i>n</i>	<i>NIEMG_{0n}</i>	<i>B_n</i>	<i>T_{n2}</i>
L ₃ -L ₄	0	0.261	0.15	10
L ₄ -L ₅	0	0.180	0.2	10
L ₅ -L ₆	0	0.338	0.14	10

NIEMG During 7-h Recovery $NIEMG(t) = E(1 - e^{-t/T_3}) + t B e^{-t/T_2} + C(t - T_d) e^{-(t-T_d)/T_4} + NIEMG_0$								
Level	<i>E</i>	<i>T₃</i> , min	<i>B</i>	<i>T₂</i> , min	<i>C</i>	<i>T₄</i> , min	<i>T_d</i> , min	<i>NIEMG₀</i>
L ₃ -L ₄	0.784	120	0.14	13	0.0025	200	275	0.216
L ₄ -L ₅	0.835	45	0.2	12	0.003	190	200	0.165
L ₅ -L ₆	0.687	64	0.16	13	0.004	320	200	0.313

Displacement During Work Period $Disp(t) = D_{0n} + D_{Ln} \{1 - e^{-(t-n(T_W+T_R))/T_{n5}}\}$				
<i>n</i>	<i>D_{0n}</i> , mm	<i>D_{Ln}</i> , mm	<i>T_{n5}</i> , min	<i>r</i> ²
0	13.014	5.525	7.5	0.939
1	18.269	2.720	8	0.870

Displacement During Rest Period $Disp(t) = D_{0n} + R_n + (D_{Ln} - R_n) e^{-(t-[(n+1)T_W+nT_R])/T_{n6}}$				
<i>n</i>	<i>D_{0n}</i> , mm	<i>D_{Ln}</i> , mm	<i>R_n</i> , mm	<i>T_{n6}</i> , min
0	13.014	5.525	5.255	2

Displacement During 7-h Recovery $Disp(t) = D_0 + R + (D_L - R) e^{-t/T_6}$				
<i>D₀</i> , mm	<i>D_L</i> , mm	<i>R</i> , mm	<i>T₆</i> , min	<i>r</i> ²
13.014	9.067	3.144	15	0.892

See METHODS for definition of terms.

review, see Ref. 26). The models fitted to experimental data show a marked decrease in the time constant T_{n1} between the two 30-min loading periods. This pattern was consistent in all of the three different groups for any of the three lumbar levels, indicating a faster EMG decrease as time progressed.

During the first 10 min of the 7 h of recovery (Fig. 5), a sharp peak of the NIEMG was observed in all of the three experimental groups (20, 40, and 60 N). This EMG pattern has already been shown (21, 26, 27, 31) and has been referred to as initial hyperexcitability. This is promoted by an increased reflex activation of the muscles that takes place to protect the already strained viscoelastic tissues from further injuries. The values obtained for the peak of the initial hyperexcitability were close or higher than the 1.0 preload value (0.84, 0.87, and 1.09 for the 20-, 40-, and the 60-N groups, respectively), and no significant difference was observed among the three different loads (20, 40, and 60 N). The modeling results showed a

general trend toward small increases in peak initial hyperexcitability with increasing loads. The constant B , governing the amplitude of the initial hyperexcitability, ranged between the values of 0.06 and 0.1 in the 20-N preparations, between 0.09 and 0.12 in the 40-N group, and between 0.14 and 0.2 in the preparations subjected to 60 N. The time constant T_2 decreased from a mean value of 16 min (20-N load) to mean values of 14 and 12.66 min observed in the 40- and 60-N groups, respectively. This indicates a tendency toward a faster rise of the initial hyperexcitability as the load increased, suggesting that a faster and stronger muscle response was required to protect the ligaments.

These results partially differ from what was previously observed. In a recent work (21) in which the impact of load magnitude on cumulative low back disorder was investigated, a significant difference of the initial hyperexcitability was found among different loads (20, 40, and 60 N), and the peak of the initial hyperexcitability was, in all cases, below the 1.0 preload value. In that study, the cumulative loading period was of the same length as in the present study (60 min), but the work-to-rest ratio was 1:1 (10-min work and 10-min rest). This suggests that the load magnitude impacts on the initial hyperexcitability component of neuromuscular disorder magnitude, yet does not seem to be the primary factor for cumulative low back disorder development when the static load duration is prolonged. This implies that the different work-to-rest ratio duration can generate a greater impact on the onset of a cumulative low back disorder.

After the initial hyperexcitability, the NIEMG gradually recovered during the first 2 h of rest. Afterward, the presence of the delayed hyperexcitability was observed in all of the three different groups. The delayed hyperexcitability is the neuromuscular response to the microdamage and acute inflammation in the viscoelastic tissues (11, 12, 26, 27, 34). An inflammatory reaction is initiated in the first hours after a tissue injury is induced and continues to increase in time (Refs. 12 and 20, among others) as the circulatory system continues to deposit inflammatory agents in the affected tissues (22). This results in delayed symptoms manifesting themselves over time, e.g., the morning after, as was termed before (27). It has been demonstrated that this response is more pronounced as a function of the time of exposure to a certain exercise (11, 12), and this seems to be confirmed by the results obtained in the present study.

The delayed hyperexcitability was characterized by a gradual NIEMG increase that reached the 1.0 initial value after 4 h of recovery in the groups subjected to 20 and 40 N and 2 h after the recovery phase started in the preparations subjected to 60 N. The modeling results showed that the time constant T_4 progressively became shorter as the load magnitude increased, ranging from the mean value of 290 min (20-N group) to the mean values of 275 and 237 min observed in the 40- and 60-N groups, respectively. The time constant T_d increased from 260 min (20-N group) to 290 min (40-N group), whereas it was shorter in the group subjected to 60 N, where T_d was 225 min. Similar to what was observed for the initial hyperexcitability, only a tendency toward an earlier and faster response of the neuromuscular system was found at the higher load (60 N), as it was not supported by the statistical analysis. In our laboratory's previous work, the delayed hyperexcitability turned out to be closely related, either to the load magnitude (21) or to the

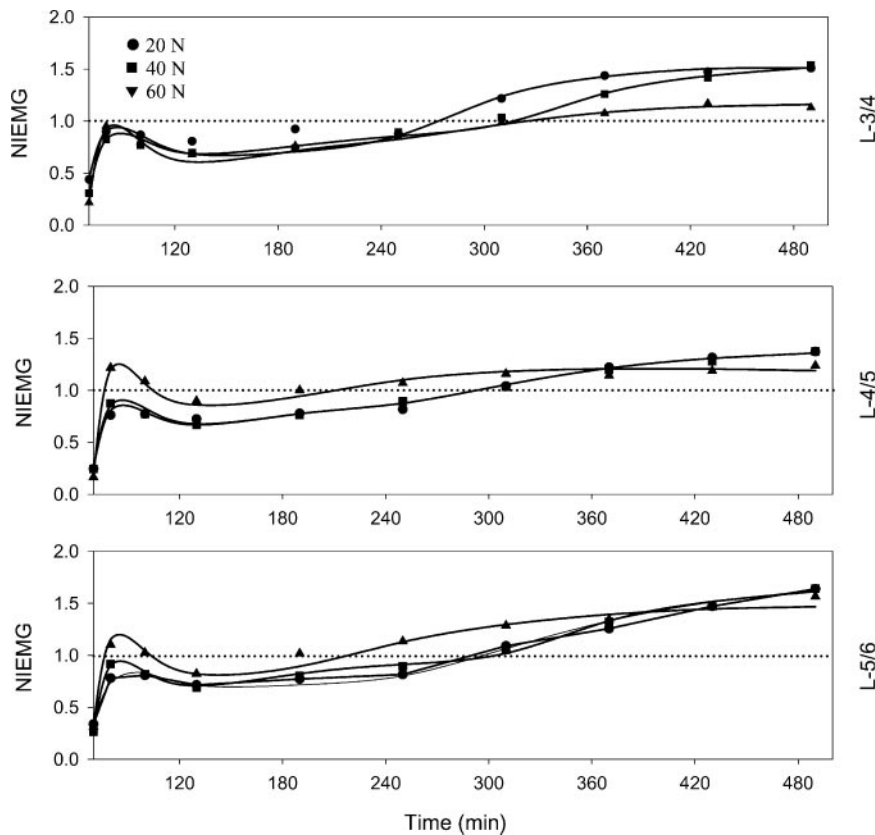


Fig. 5. Mean NIEMG data and the developed models for the 7-h recovery period are superimposed for 20-, 40-, and 60-N loads.

overall time of loading (22). Once again, it has to be pointed out that, in the first study (21), a 1:1 work-to-rest ratio was used, and this might have allowed a partial recovery of the creep and NIEMG, at least at the lower loads applied to the lumbar spine (20 and 40 N). When a 1:1 work-to-rest duration ratio was given (31), similar results were obtained as well, although, in that study, the feline preparations were subjected to the load of 40 N only, and a cumulative 30-min loading period was adopted.

In accordance with the results obtained in the present study, at least two relevant observations can be addressed. First, the major role in promoting a neuromuscular disorder was represented by the longer continuous duration over which a load was sustained. Therefore, the load duration is established as the primary risk factor, in association with the load magnitude that, at least in this experimental design, should be considered of secondary importance. Second, and this appears to be the key point, the 3:1 work-to-rest duration ratio resulted in full neuromuscular disorder, regardless of the load magnitude, which had only a relatively moderate impact on the severity of the disorder.

The results obtained in the present study confirm, from a physiological standpoint, what is already reported by the epidemiology. It seems that the creep that developed within the viscoelastic tissues as a consequence of the long static load applied to the lumbar spine causes microdamage and triggers inflammation (20, 34). This process requires time as the inflammatory cells are released via the bloodstream to the injured tissues (11). Over time, the migrations of neutrophils and cytokines into the collagenous tissues leads to a full inflammatory condition (26). From this point on, the neuromuscular

pattern observed might reflect a protective response to the microdamage provoked in the viscoelastic tissue or to the inflammatory process, preventing further damage while healing takes place. The delayed hyperexcitability component observed in all of the preparations represents the physiological response to such a phenomenon. It appears that, if the lumbar spine is subjected to a static load, which is prolonged over time, an overnight rest would not be enough to recover from the microdamage promoted. It is indeed conceivable that a complete recovery from the early signs of the neuromuscular disorder, caused presumably, would take more than 2 days. As a consequence, without sufficient recovery, the residual creep and acute inflammation may accumulate from day to day and finally mature to chronic inflammation, triggering a CTD.

The overall response to the application of static flexion was considered a disorder. Clinically, spasms are considered as a symptom to tissue damage, as was also confirmed experimentally (18). Hyperexcitability, as measured by elevated EMG, is also established as a response to low back disorder (5, 7, 9, 14, 23). It is associated with increased muscular contraction and stiffness, which, in turn, limit the normal range of motion. Therefore, despite the expectation that the response is acute and will be resolved in 1–3 days, clinically, it constitutes a neuromuscular disorder.

Generalization of data collected from animals to humans should be considered and reconfirmed very carefully while making the necessary adjustment, calibrations, and modifications. Our early work in this direction confirmed that a reflex from the supraspinous ligament to the multifidi also exists in humans (30). More recently, we also demonstrated that human subjects performing 10 min of static lumbar flexion exhibit

spasms during the flexion and profound changes in muscular activity postflexion (25). Furthermore, in an attempt to extend the findings to ligaments of other joints, we found that 10 min of static loading of the anterior cruciate ligament of humans also resulted in spasms and postloading hyperexcitability of the quadriceps (1). Ongoing work with human subjects also confirms that cyclic loading of the spinal and knee ligaments elicits a neuromuscular disorder. Overall, human subjects seem to respond in a similar mode to that of the feline model when subjected to static and cyclic loading of ligaments. So far, the insight gained from the feline model has proved to be valuable for understanding human responses to similar conditions.

From the clinical standpoint, low back patients present with pain, posterior muscle stiffness, and limited range of motion. Muscle stiffness and the associated elevated EMG are widely reported in the literature (Refs. 5, 7, 9, 14, 23, and others). It is evident that the increased EMG (or hyperexcitability) that we observed represents the stiffness associated with higher than normal muscular force response and the resulting limited range of motion. In essence, the responses observed in the feline model have some parallelism to clinically established symptoms as well.

It should be noted that the responses observed in this report represent an acute condition that is expected to be resolved in 1–3 days (based on model predictions). CTD, however, will require daily repetition of the static flexion for a long period in order for the condition to be transformed from acute to chronic.

Finally, our laboratory's recent findings (26) confirm that the S-shaped hook did not elicit an artifactual response due to application of force to a localized area of the ligament. Neutrophils of similar concentration were observed throughout the L₄-L₅ supraspinous ligament and in the ligaments of one level above and below, confirming that the overall tissue strain elicited by the lumbar flexion was the source of the response while excluding the localized strain due to the hook. The fact that lumbar flexion was elicited by loading the L₄-L₅ supraspinous ligament was also confirmed by X-ray records (33). An isolated control group further demonstrated that the mere presence of the S-shaped hook was not the source of the disorder (33). We can reasonably conclude that the data presented are the responses of spinal viscoelastic tissues to static flexion.

In conclusion, two 30-min static loads spaced by a 10-min interval elicited a cumulative low back disorder that did not recover after 7 h of rest. Although the impact of load magnitude cannot be neglected, this was overwhelmed by the effect of longer static loading duration and its distribution (work-to-rest ratio). It can be reasonably argued that the combined effect of load magnitude, duration of loading, number of repetitions, and work-to-rest duration ratio as a whole should be considered as risk factors in CTD development. An optimal dose-to-duration ratio is yet to be determined to limit, attenuate, or prevent the adverse effects of static load on the lumbar spine (6).

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