ABSTRACT: Epidemiologic studies indicate that repetitive (cyclic) occupational activities lead to a cumulative trauma disorder (CTD), and the frequency or velocity of the movement is one of the risk factors. Experimental neurophysiological evidence to confirm the epidemiology is not available. The response of the multifidus muscles to cyclic loading in anterior lumbar flexion–extension was assessed to test the hypothesis that high-frequency loading may induce an acute neuromuscular disorder leading to CTD. Two groups of feline preparations were subjected to cyclic loading with a peak of 20 N: one at 0.25 Hz and the second at 0.5 Hz, with an equal number of cycles. Electromyogram (EMG), lumbar displacement and load were recorded throughout the loading periods and during single-cycle tests over a 7-hour rest period following the load–rest sessions. A model was developed to quantify the creep and neuromuscular responses, and analysis of variance (ANOVA) was applied to assess significance of the results. The group exposed to 0.5 Hz exhibited spontaneous spasms followed by sustained spasms during the loading periods. During the 7-hour recovery period, a significant ($P < 0.001$) delayed hyperexcitability as well as sustained spasms of the multifidi were present in the last 5 hours, confirming a significant ($P < 0.024$ to $P < 0.042$) acute neuromuscular disorder. High-frequency cyclic loading of the lumbar spine may trigger a severe acute neuromuscular disorder, as evidenced by the sustained spasms and delayed hyperexcitability, and should be considered as a risk factor. We suggest that workers avoid high-frequency exposure to cyclic activity in order to prevent the development of cumulative trauma disorder.
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FREQUENCY OF CYCLIC LUMBAR LOADING IS A RISK FACTOR FOR CUMULATIVE TRAUMA DISORDER

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Cumulative trauma disorder (CTD) is a musculoskeletal disorder common to individuals engaged in repetitive or cyclic activities over prolonged periods.[8,32] Typical examples are loading/unloading boxes, repeating the same movement on assembly lines, cyclic drill pressing, etc. Symptoms include pain, weakness, and limited range of motion in the afflicted joint and muscular stiffness/spasms in the surrounding area. Epidemiological studies have shown a statistical correlation between CTD and load magnitudes, number of repetitions, duration of activity, and the velocity or frequency at which such movements are performed.[5,28] To date, emerging biomechanical and neurophysiological data confirm that high cyclic load magnitudes,[9] number of repetitions,[16] and insufficient rest between work periods[6] elicit an acute neuromuscular disorder indicative of acute inflammation[29] in the viscoelastic tissues (ligaments, facet capsules, disks, etc.) of the lumbar spine of the feline model. Additional studies have shown that such an acute disorder is present in healthy human subjects exposed to cyclic lumbar activity[1,11,18,19] and to cyclic knee loading.[26]

Lu et al.[12] provided preliminary evidence confirming that cyclic movement performed at high frequency may elicit significantly more creep in the lumbar viscoelastic tissues. Spinal models also pointed out that lumbar motion performed at high

Abbreviations: ANOVA, analysis of variance; CTD, cumulative trauma disorder; EMG, electromyogram; L-3/4, lumbar third to fourth vertebrae joint; L-4/5, lumbar fourth to fifth vertebrae joint; L-5/6, lumbar fifth to sixth vertebrae joint; MTS, material testing system machine; NIEMG, normalized integrated electromyogram

Key words: cumulative trauma disorder; cyclic; disorder; lumbar; multifidus; spine

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velocities associated with high cyclic frequency may develop high loads in the various tissues and may lead to risk of injury.[3,13,14,22] It is still necessary to experimentally confirm, from the biomechanical and neurophysiological standpoint, that increased frequency of cyclic lumbar motion produces an acute neuromuscular disorder.

It is hypothesized that cyclic lumbar loading at low loads, low frequency with sufficient rest, and low number of repetitions will not trigger a neuromuscular disorder, whereas increasing the frequency while keeping the overall number of loading cycles constant will trigger an acute disorder.

The expected conclusions from such an investigation will experimentally confirm the epidemiology, define the etiology of CTD relevant to frequency as a potential risk factor, and will provide guidelines for optimal work schedules to prevent occupational disorders.

METHODS

Preparation. Thirteen cats (3.8 ± 0.39 kg) were anesthetized intravenously with α-chloralose (60 mg/kg) in a protocol approved by the institutional animal care and use committee at our institution. An incision was made from the thoracic to sacral level and retracted laterally to expose the dorsolumbar fascia. A stainless-steel, S-shaped hook was applied around the supraspinous ligament between L-4 and L-5. The preparation was placed on a rigid stainless-steel frame with external fixation that isolated the lumbar spine from the thoracic and sacral levels. A saline-soaked gauze pad was placed over the exposed area to prevent tissue drying. Two groups were assessed: one at a cyclic frequency of 0.25 Hz, subjected to a 20-N peak load ($N = 6$), and another group, also with 20-N peak load ($N = 7$), but at a frequency of 0.5 Hz.

Instrumentation. With the specimen positioned prone on the stainless-steel frame, external fixtures were placed on the posterior processes of L-1 and L-7 to isolate the specimen. The external fixtures were placed on the ends of the lumbar vertebrae to prevent interaction with the thoracic, sacral, and pelvic sections of the spine, but not to limit lumbar motion.

Three pairs of electromyographic (EMG) fine-wire electrodes were inserted 5–6 mm to the right of the midline into the multifidus muscles at L-3/4, L-4/5, and L-5/6, respectively. A ground electrode was placed in the non-related gluteus muscle. Each electrode pair constituted the input to a differential amplifier having a 110-dB common mode rejection ratio, a gain capacity of up to 200,000, and a bandpass filter of 6–500 Hz. The EMG response was constantly monitored on oscilloscopes and recorded on a computer at a sampling rate of 1000 Hz. The actual gain for each of the three EMG channels was ×50.

A stainless-steel, S-shaped hook was inserted around the supraspinous ligament between L-4 and L-5 and connected to the vertical actuator of a material testing system (MTS; Bionix 858; MTS, Inc., Minneapolis, Minnesota). The load was applied by the MTS actuator with a computer-controlled loading system operated in load-control mode. Vertical displacement of the actuator and the load cell output were also sampled into the computer along with the EMG data.

Protocol. A cyclic tensile vertical load of 20 N was applied to the lumbar spine via the stainless-steel hook between L-4 and L-5 at a frequency of 0.25 Hz for a period of 10 minutes. The 20-N force was determined in a previous study to be at the low end of the physiological range, just above the threshold of reflex EMG responses in the muscles.[31] Further, 0.25 Hz was chosen as the preferred frequency in a survey of healthy young human subjects requested to perform full anterior flexion followed by extension to normal posture at a personally selected comfortable rate. Next, the load was withdrawn, and the preparation was subjected to a rest period of 10 minutes. Six such cyclic load–rest periods were applied for a total of 2 hours, for a cumulative loading period of 60 minutes.

During the 7-hour rest/recovery period following the cyclic load–rest period, single-cycle tests were performed to assess vertical displacement along with residual creep and EMG recovery. Tests were applied after 10, 20, and 30 minutes of rest, and each hour subsequently yielded nine data points. Each test consisted of a single cycle to 20-N peak and 0.25 Hz. The tests were triggered by the computer at set intervals. Vertical tension was fully released between the recovery tests.

The same protocol, but with a 5-minute load followed by 5-minute rest repeated six times with a peak load of 20 N at 0.5 Hz, was used for comparison with the 0.25-Hz paradigm. Similar single test cycles at 20 N and 0.5 Hz were taken during the 7-hour recovery. Because the number of repetitions was shown to be a significant risk factor,[16] the number of load cycles during the 5 minutes at 0.5 Hz was equivalent to the number of cycles over 10 minutes at 0.25 Hz. In order to further avoid the possibility of insufficient rest, the 1:1 ratio of work to rest for low load was observed, as shown by Hoops et al.[6] In their study, it was shown that if the work:rest ratio was kept

at 1:1, the effect of longer or shorter periods of loading had no impact on development of an acute neuromuscular disorder for a range of work periods up to 1 hour. Therefore, the outcome of the present study should be free from confounding effects of rest period differences and could be considered as the effect of higher cyclic frequency.

Analysis. Cyclic loading, vertical displacement, and EMG response were recorded throughout the experiment. After the first loading cycle of the first 5- or 10-minute phase, samples were recorded every 20 seconds during each 5- or 10-minute cyclic loading session and for each single cycle test during the recovery period.

Each EMG sample was integrated over the respective cycle's window and normalized with respect to the value of the first cycle of the first 5- or 10-minute loading period in each data set. The normalized integrated EMG (NIEMG) of each specimen was classified into its respective load and lumbar level. The mean (± SD) was plotted as three different sets: NIEMG, load, and displacement vs. time.

Model. The model being utilized is from previous work based on 10-minute static loading and 10-minute rest periods for a 2-hour period and then a 7-hour recovery period.[6,9,16,30] In this study, the model equations are analyzed based on cyclic loading behavior where the equations represent two separate entities: one is the cyclic loading period, and the other is based on the recovery period. The model for the loading session of NIEMG can be described as follows:

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

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

The model for long-term recovery is also based on exponential decay. It is described as follows:

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

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

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

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

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

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

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

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

In a similar manner, an equation describing residual displacement (viscoelastic creep) is defined throughout the work periods as follows:

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

T_{n5}, δ_{0n}, and δ_{Ln} are variable from one work–rest session to the next. Residual recovery from viscoelastic displacement is expressed as follows:

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

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

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

Best-fit models were generated after the calculation of the mean and standard deviation using the Marquardt–Levenberg nonlinear regression algorithm. In some cases, sequential recursive iteration was applied manually to optimize the regression.

Statistical Analysis. A two-way mixed model analysis of variance (ANOVA) was used to analyze the NIEMG from each lumbar level. Independent variables for the test included cyclic frequency (0.25 Hz, 0.5 Hz) and time elapsed during the recovery period (10, 30, 60 minutes, etc.). Dependent variables for the test included NIEMG from each lumbar level (L-3/4, L-4/5, L-5/6). Upon finding a significant effect of time, the changes were analyzed for each combination of cyclic frequency and lumbar level using a one-way mixed-model ANOVA, and post hoc comparisons were performed using Student's *t*-test. Level of significance was set at $P = 0.05$ for all tests. Before performing the analysis, NIEMG data from each lumbar level were logarithmically transformed to obtain a normal distribution.

RESULTS

Typical recordings of EMG, displacement, and load from one specimen subjected to 20 N at 0.25 Hz and one at 0.5 Hz are shown in Figure 1A and 1B, respectively. The EMG patterns in Figure 1A suggest exponential decay during the cyclic work sessions, with partial recovery between rest sessions. In Figure 1B, sustained, large-amplitude spasms appear throughout, because the overall EMG response decreased during the cyclic loading. Pronounced multifidus spasms also occurred in the later hours of the recovery for the 0.5-Hz category (Fig. 1B). At the end of the cyclic work period, creep had accumulated substantially, without full recuperation after the 7-hour recovery period.

Figure 2A and B displays the mean ± SD data for the two frequency categories. Figure 3 provides the NIEMG responses of the two frequencies during the recovery period.

Displacement and Creep. The group subjected to the 20-N cyclic load at 0.25 Hz (Fig. 2A) had an initial (at the beginning of the first loading session) mean displacement of 3.50 mm, with an increase to 7.14 mm at the end of the first 10-minute loading period. The associated mean creep was 104% and the first rest period elicited a decrease in mean creep to 57.1%. The creep accumulated over the 2-hour cyclic work–rest session and resulted in a final value of 177.4%. At the end of the 7-hour recovery period, the mean creep decreased to 65.1%.

The group subjected to cyclic loading at 0.5 Hz (shown in Fig. 2B) had a mean initial displacement of 4.09 mm and, at the end of the first 5-minute loading period, it reached 6.57 mm, corresponding to a creep value of 60.6%. The first 5-minute rest period allowed the creep to recover to 32.5%. Creep continued to accumulate over the following five loading periods with a final creep value of 103.2%. At the end of the 7-hour rest following the six load–rest periods, the residual creep was 52.3%.

NIEMG. Mean NIEMG for the 0.25-Hz group displayed decreasing values to 0.526, 0.511, and 0.454 after the first 10-minute cyclic work session in the L-3/4, L-4/5, and L-5/6 multifidus muscles, respectively. The first 10 minutes of rest displayed recovery to 0.829, 0.814, and 0.784 for the respective levels. The succession of work–rest sessions resulted in a further decrease in NIEMG to final values of 0.408, 0.399, and 0.425, respectively. An increase in NIEMG was observed after the last work–rest period, followed by a slight decrease during the first hour. A slow exponential increase was evident during the remaining hours of recovery. Final values after the 7-hour recovery period were 0.861, 0.843, and 0.897 for the respective multifidus levels. Full recovery of the NIEMG was not seen in any of the preparations.

The mean NIEMG for the 0.5-Hz group decreased during the first 5-minute loading period to 0.620, 0.538, and 0.624 for L-3/4, L-4/5, and L-5/6, respectively. Due to the consistent sustained spasms of large amplitude in the second through sixth loading sessions, the NIEMG did not show the expected exponential decrease during loading, nor the partial recovery during the rest period, yielding an erratic and nonrepresentative pattern, which was observed in the previous studies. The exceptionally high amplitude and sustained spasms in each of the seven preparations of this category represent an important finding (see Discussion). During the 7-hour recovery period, a slight increase was observed at the end of the 30-minute post-loading period, followed by a decrease to the end of the first hour. The mean NIEMG increased thereafter, reaching a peak at 1.23 at the fifth hour for the L-3/4 multifidi, a peak of 1.29 at the sixth hour for the L-4/5, and 1.47 at the sixth hour for the L-5/6 level. The peak NIEMG values represent delayed hyperexcitability of 23%, 29%, and 47% in the L-3/4, L-4/5, and L-5/6 multifidi, respectively.

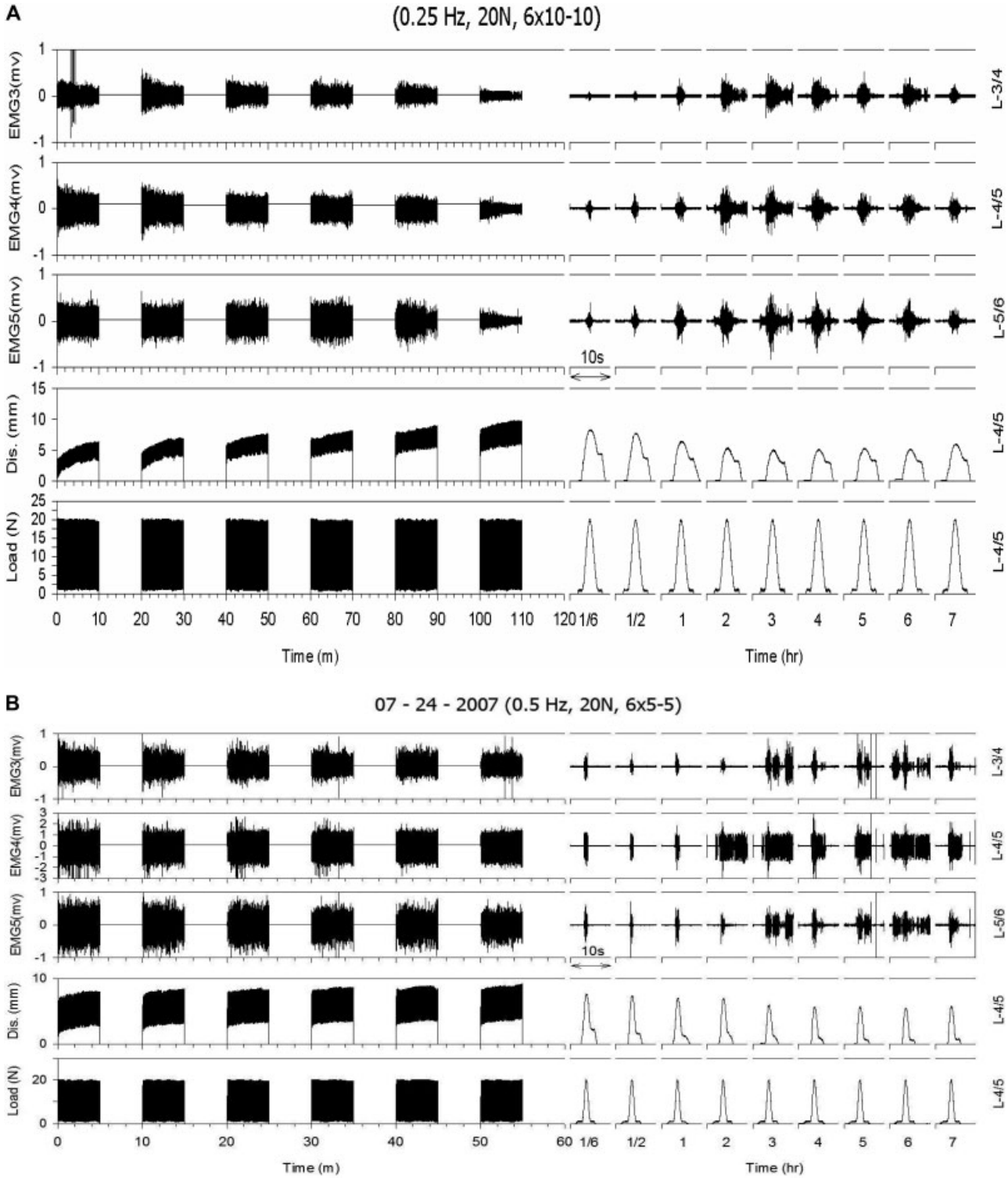

FIGURE 1. Typical EMG from L-3/4, L-4/5, and L-5/6, with displacement and tension applied to the lumbar spine for six cycles of 10 minutes of work followed by 10 minutes of rest at 0.25 Hz **(A)** and six cycles of 5 of minutes work followed by 5 minutes of rest at the same load of 20 N at 0.5 Hz. Both **(A)** and **(B)** also depict the nine test cycles during the 7-hour recovery period post-loading. Note a typical random spasm in the first loading session of L-3/4 in **(A)** and the sustained spasms with increasing amplitude in the second to sixth sessions of L-4/5 and L-5/6 in **(B)**.

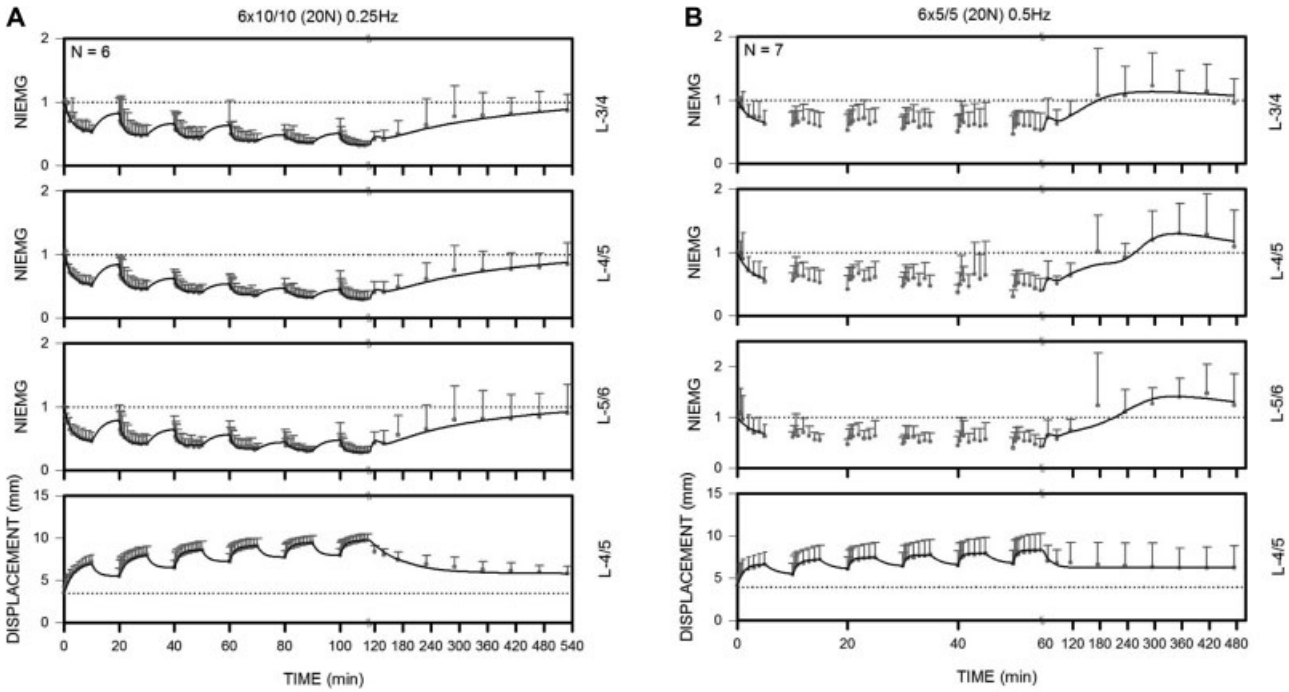

FIGURE 2. The compounded data for NIEMG and displacement is shown (mean ± SD) for the set loaded at 0.25 Hz **(A)** and for 0.5 Hz **(B)**.

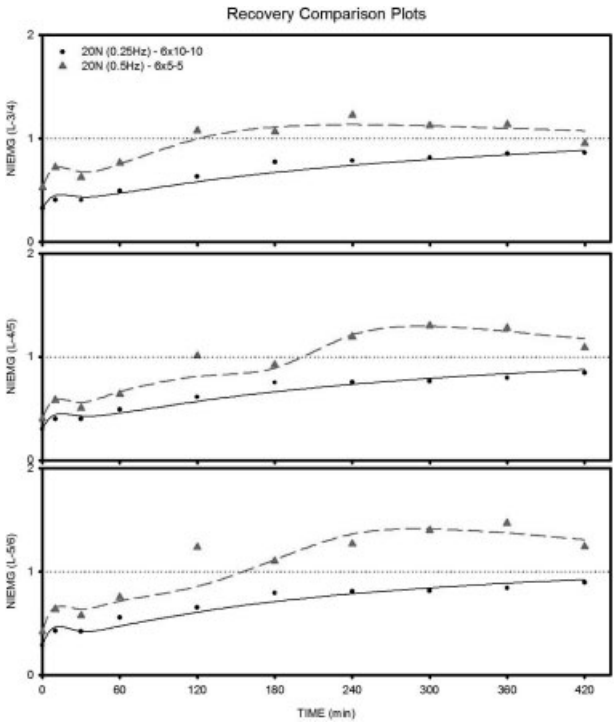

FIGURE 3. The NIEMG patterns and associated models of the three lumbar levels during the 7-hour recovery are shown for the 0.25-Hz and 0.5-Hz groups. The two curves at each level are significantly different from each other.

The Model. The best-fit models derived for the compounded data of the 0.25-Hz and the 0.5-Hz categories are superimposed on the mean ± SD of the NIEMG and displacement shown in Figure 2A and B. The R^2-values for the models ranged from 0.7910 to 0.9938, and were mostly >0.9.

Statistical Analysis. The statistical analysis confirmed that each NIEMG recovery curve was a function of time ($P < 0.001$) and that the corresponding curves of the NIEMG of the same lumbar level at 0.25 Hz and 0.5 Hz were significantly different, with $P < 0.024$ for L-3/4, $P < 0.042$ for L-4/5, and $P < 0.02$ for L-5/6. Interaction of time and frequency were not present with $P < 0.284$, $P < 0.545$, and $P < 0.522$ for L-3/4, L-4/5, and L-5/6, respectively.

DISCUSSION

The most important finding from this investigation consists of the evidence that doubling the frequency of a passive cyclic flexion–extension of the lumbar spine from 0.25 Hz to 0.5 Hz elicits delayed hyperexcitability. This is indicative of an inflammation in the viscoelastic tissues some 4–5 hours post-activity. The observation that sustained and overwhelming (as opposed to random) spasms were present throughout most of the cyclic load–rest period in the 0.5-Hz group further supports the possibility that

substantial damage to the viscoelastic tissues had already occurred due to the high velocity of their deformation. Furthermore, although the 0.5-Hz group was subjected to the exact same number of cycles applied to the 0.25-Hz group, the cumulative work time was only 30 minutes, half as much that of the 0.25-Hz group. The applied load was also very light. Despite such substantially more favorable exposure, a delayed neuromuscular disorder developed during the post-loading period as well as overwhelming sustained spasms throughout, underscoring the effect of higher frequency. In light of the findings obtained in previous projects,[6,9,16] one can surmise that high-frequency cyclic loading of the lumbar spine is probably the most deleterious risk factor for development of an acute neuromuscular disorder, which with time may lead, if further aggravated, to CTD.

Our earlier work[29] identified excessive spasms and delayed hyperexcitability with a sharp increase (100-fold) in neutrophil density in the affected ligaments 2–3 hours post-loading. Histologic change and hyperexcitability seem to be present only when damaging stimuli are applied. The presence of elevated neutrophil density indicates inflammation in its acute phase. Clinical experience points out that acute inflammatory conditions exposed to further physical activity over time yield chronic inflammation and degeneration of the ligamentous tissues.[10,25]

Spontaneous spasms, such as those seen in Figure 1A (top left), are an indication of damage occurring to tissues.[23] Conversely, sustained/ongoing spasms (such as those seen in Fig. 1B: L-4/5, first five sets of loading) producing long-term stiffness in a joint are known to exist in low-back-pain patients with already damaged tissues (such as herniated disk, facet capsule impingement, ligament sprain, etc.).[2,4,7,15,24,27] The first 5-minute loading session applied to the 0.5-Hz group exhibited mostly spontaneous spasms while still following the exponentially decreasing NIEMG. It is reasonable to assume that the higher frequency of cyclic loading caused significant damage to the viscoelastic tissue over the first 5-minute loading.[17,21] Consecutive 5-minute loading sessions exhibited mostly ongoing, sustained spasms, which completely masked the expected exponential decrease in the NIEMG and mostly increased EMG during the loading (see Fig. 1B: L-4/5 and L-5/6). This probably produced stiffening of the joints to protect the injured tissues from further negative effects of the fast cyclic loading. Indeed, the displacement and creep data support this interpretation. The mean creep at the end of the initial cyclic loading session was 104% for the 0.25-Hz group and only 60.6% for the 0.5-Hz group. Similarly, the final creep at the end of six sessions of load–rest was 177% for the 0.25-Hz group and only 103.2% for the 0.5-Hz group. Furthermore, residual creep at the end of the 7-hour rest was 65.1% and only 52.3% for the 0.25-Hz and 0.5-Hz groups, respectively.

It was expected that creep for the 0.5-Hz group would be much higher, as observed in a previous study,[12] yet the sustained spasms stiffened the lumbar spine. This offered significantly higher resistance to the load and yielded smaller displacement and associated creep. This observation was also consistent with previous work. Each time a risk factor (such as high load, high number of repetitions, too little in-between rest, etc.) elicited a neuromuscular disorder, the creep values were substantially lower than values corresponding to paradigms with such factors at a mild or moderate level and without triggering a delayed disorder.[6,9,16] Spasms seem to offer protection to the damaged tissues.

Spasms were also observed during the 7-hour recovery period of the 0.5-Hz group. From Figure 1B, one can see that, from the second to the seventh hour of the recovery period, spasms were not only triggered by the test cycles or lingered after the cycle, they were present even before the test cycles were applied (as seen in the sixth test cycle during the recovery period in Fig. 1B).

Overall, sustained spasms were a dominant component in the 0.5-Hz group, during the load–rest period and during the recovery period. Damage to the viscoelastic tissue from the high loading frequency of 0.5 Hz apparently shifted the motor control mode from the routine, reflexive response to a mode of damage control. This was manifested by the application of sustained stiffness to the lumbar spine in order to shield and protect the damaged tissue from further exposure to the negative effects of the high-frequency load. Under normal conditions, the reflex response of the multifidi expresses load sharing between ligaments and muscles while maintaining intervertebral stability. Under such motor control strategy, as the tension in the ligaments exceeds a certain level, the muscles are triggered reflexively in order to keep stability. The shift from a motor control scheme to a damage control scheme occurs when the ligaments are damaged, and the musculature assumes a major role in protecting them as well as preventing additional damage by stiffening the joint.

The peak load, number of repetitions, and load: rest ratio were the same, and the overall duration of loading was favorable: only half for the 0.5-Hz group relative to the 0.25-Hz group. The significantly deleterious response underscores the magnitude of the

effect of the only variable, higher frequency. It can be concluded that high frequency of cyclic loading is probably more deleterious than other risk factors, as it inflicts tissue damage even at very low loads and short loading durations. High frequency, therefore, should be avoided during cyclic work, as it may lead to severe low-back disorders.

Based on the results of this investigation, the following conclusions are proposed:

1. Cyclic lumbar flexion at high frequency is a risk factor for an acute neuromuscular disorder, as it produces substantial hyperexcitability 5–6 hours after load–rest activity.
2. From the pattern of the spasms seen in this investigation, substantial injury was developing in the viscoelastic tissues within a short period of exposure to fast cyclic loading. Thereafter, the musculature shifted function to provide continuous stiffness to the spine to prevent further injury to the tissues.
3. Fast cyclic loading emerges as the most deleterious risk factor (when compared to high load magnitudes, short rest periods, large number of repetitions, and long exposure periods), as it elicits a complex disorder with components during the loading and afterwards even at a low load magnitude, a short loading duration, an ample rest, and a low number of repetitions. Fast cyclic loading, therefore, should be avoided as much as possible when performing occupational or sports activities to prevent tissue damage and the development of an acute neuromuscular disorder that may further evolve into a chronic disorder with continuous exposure of the tissues to activity.

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