ABSTRACT: Epidemiological data suggest that prolonged exposure to cyclic lumbar flexion elicits a chronic neuromuscular disorder and disability in workers. This study provides a physiological and biomechanical assessment of various repetitions of cyclic lumbar flexion sessions as a risk factor for development of an acute neuromuscular disorder. An in vivo feline model was subjected to 10 minutes of cyclic (0.25-Hz) loading, followed by a 10-minute rest period, repeated three times in one experimental group, six times in a second group, and nine times in the third group, followed by rest for 7 hours. Displacement of the lumbar viscoelastic tissue and reflex electromyographic (EMG) activity from the lumbar multifidus muscle were monitored. Creep developed and accumulated during each load/rest period and partially recovered during the subsequent rest. Loading periods were characterized by a decrease in reflex EMG activity with superimposed spasms. In the 7-hour recovery period, initial hyperexcitability was present in all groups, whereas only the six- and nine-repetition groups displayed significant delayed hyperexcitability, indicating the presence of acute inflammation. The mathematical model developed fit the data reasonably well, as the $R^2$ values were generally near 0.90. It was concluded that the resulting delayed muscular hyperexcitability constitutes an acute neuromuscular disorder associated with exposure to many repetitions of cyclic lumbar flexion. The acute disorder can become chronic if not allowed sufficient rest to resolve itself. Workers engaged in cyclic lumbar flexion (e.g., loading/unloading, assembly workers) should avoid long-term exposure in order to prevent the development of a chronic neuromuscular condition known as cumulative trauma disorder.

**HIGH-REPETITION CYCLIC LOADING IS A RISK FACTOR FOR A LUMBAR DISORDER**

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Epidemiological data have suggested that long-term exposure to repetitive (cyclic) work is a risk factor for musculoskeletal disorders.⁶,¹⁵,²¹ In the United States, these disorders have led to more disability or absenteeism from the workplace than any other group of diseases.¹⁵ The cyclic lifting associated with musculoskeletal disorder is not necessarily at extreme loads. In most cases the load lifted is well within the physiological range, but over time a cumulative effect is observed. The cumulative effect of cyclic lifting over a day can manifest itself as creep (laxity) of the viscoelastic tissues, which is caused by microdamage in the collagen fibers.⁵¹ An acute inflammation is triggered by the microdamage, representing an attempt to heal the damaged tissues.⁹ Such an inflammatory response that is not given sufficient rest for recovery before the tissues are exposed to further stress may lead, over time, to chronic inflammation that results in chronic neuromuscular disorder and disability.¹⁰,¹⁶,²⁸ This chronic condition, known as cumulative trauma disorder, is characterized by pain, weakness, reduced range of motion, and spasms/stiffness of the lumbar muscles.¹²,¹⁵,²⁰

Previous work has investigated the effect of load magnitude, load duration, and the number of repetitions of static lumbar flexion on the development...
of an acute lumbar disorder in cats. In static lumbar flexion, increasing the number of repetitions leads to an acute neuromuscular disorder that is triggered by acute inflammation and is characterized by spasms and hyperexcitability of the multifidus muscles. Specifically, a break point was observed between six and nine lumbar flexion/rest repetitions, with nine repetitions triggering an acute disorder. Experiments have investigated the effect of frequency in cyclic lumbar flexion and supported the belief that cyclic lifting may be a risk factor in humans, but the effect of increasing the number of repetitions requires study.

We hypothesized that, as the number of repetitions increases, more creep will develop as will a more intense, delayed acute neuromuscular disorder during the recovery period. Our objective was therefore to obtain biomechanical and physiological evidence of this and thereby provide experimental validation of epidemiological data.

**METHODS**

**Preparation.** Nineteen adult cats (3.88 ± 2.02 kg) were anesthetized using a single intraperitoneal injection of chloralose (60 mg/kg) in a protocol approved by our institutional animal care and use committee. The skin over the lumbar spine was disected from the thoracic to the sacral level and allowed to retract laterally to reveal the dorsolumbar fascia. The preparation was then placed in a rigid stainless-steel frame in which the lumbar region was isolated with external fixation. Drying of the fascia or other exposed tissue was prevented by placing a gauze pad soaked in saline over the incision. Three groups were studied: the first group was subjected to three load/rest sessions; the second to six sessions; and the third to nine sessions.

**Instrumentation.** Three pairs of stainless-steel fine-wire electromyographic (EMG) electrodes were inserted through hypodermic needles into the right sides of the multifidus muscles of the L3–4, L4–5, and L5–6 regions, positioned 5–6 mm from the midline with an interelectrode distance of 3–4 mm. The wire electrodes were insulated with the exception of a 1-mm exposed tip. A ground electrode was inserted into the gluteus muscle. An electrode pair represented the input to a differential amplifier with a 110-dB common mode rejection ratio, a gain capability of up to 200,000, and a bandpass filter of 6–500 Hz. The EMG response from each channel was monitored by oscilloscope and stored in a computer at a sampling rate of 1000 Hz. Whereas intramuscular electrodes normally record EMG with frequency components over 500 Hz, power spectral analysis demonstrated that, for the slow-twitch multifidi at the moderate reflex force levels observed in this study, less than 9% of the power is observed at over 500 Hz, resulting in little or no loss of signal.

An S-shaped stainless-steel hook was inserted around the L4 and L5 section of the supraspinous ligament and connected to the vertical actuator of a material testing system (MTS; Model 858; Bionix, Minneapolis, Minnesota). The load was applied by the MTS actuator with a computer-controlled loading system operated in a load-control mode. The vertical displacement of the actuator and the load-cell output incorporated in it were also sampled into the computer along with the EMG data.

The lumbar spine was isolated, with external fixation at the L1 spinal process and at the L7 spinal process, to limit the elicited flexion to the lumbar region and prevent thoracic and sacral interaction. The purpose of the fixation, however, was not to prevent any motion.

**Protocol.** The stainless-steel hook inserted around the L4 and L5 supraspinous ligament was pulled by the MTS actuator from a resting position to a load of 40 N (which was determined in a previous study to be in the middle of the physiological range) at a rate of 0.25 Hz for a 10-minute period. Afterward the load was removed, allowing for a 10-minute rest period. Three such load/rest periods were applied to the first group to total 30 minutes of cyclic loading. Six such load/rest periods were applied to the second group and nine to the third group to total 60 and 90 minutes of cyclic loading, respectively. During 7 hours of rest following the completion of the load/rest session, 4-second tests were performed to assess displacement (and creep) and EMG recovery. Tests were applied after 10, 30, and 60 minutes of rest, and every hour thereafter. Each 4-second test consisted of a single cycle of a 40-N peak load pulled at 0.25 Hz (the same rate as in the load periods). The 4-second test was recorded in 12-second windows triggered by the computer at appropriate times. Between specified tests, the spine was unloaded.

**Analysis.** We sampled 4-second windows of EMG, cyclic load applied to the spine, and vertical displacement of the L4–5 supraspinous ligament at the beginning of the loading period and every 20 seconds thereafter for each 10-minute cyclic loading period, as well as for each test in the recovery period. Each EMG sample was full-wave rectified and integrated.
over the 4-second window and normalized with respect to the value obtained from the first window of the first 10-minute load period. The normalized integrated EMGs (NIEMG) of all the preparations subjected to the same number of repetitions were pooled, and the mean and standard deviation was calculated and plotted on a NIEMG vs. time plot for each of the three groups. Displacements of the respective window of all preparations subjected to the same number of repetitions were pooled, and the mean (±SD) was calculated and plotted in relation to time.

Model. The model considered is based on our previous work, in which a continuous 20-minute static load was followed by a 7-hour recovery period.19 The NIEMG during the cyclic loading period is described by eq. (1) as follows:

\[
\text{NIEMG}(t) = A e^{-t/T_1} + \text{NIEMG}_{ss}
\]

in which NIEMG_{ss} is the steady-state amplitude; A is the amplitude of the exponential component; T_1 is the time-constant of the exponential component; and t is time.

Correspondingly, the NIEMG during the long-term recovery is modeled by eq. (2):

\[
\text{NIEMG}(t) = B e^{-t/T_2} + E(1 - e^{-t/T_3})
+ C(t - T_3) e^{-t(T_3 - T_2)/T_2} + \text{NIEMG}_{ss}
\]

in which B, C, and E are the amplitudes of the three terms; \(B e^{-t/T_2}\) represents the initial hyperexcitability, which decays within 1 hour while reaching its peak in the first 10 minutes; \(C(t - T_3) e^{-t(T_3 - T_2)/T_2}\) represents the delayed hyperexcitability (initiated during the rest period, mostly after the second hour of rest, with no effect in the first 2 hours); \(E(1 - e^{-t/T_3})\) represents the steady-state recovery (slowly rising exponential throughout the rest period); T_3 is the time delay associated with initiation of the delayed hyperexcitability; and NIEMG_{ss} is the steady-state amplitude as defined in eq. (1).

In order to convert eqs. (1) and (2) to describe a series of work periods spaced by rest periods, two new components are defined: \(T_W\) is the time period over which load was applied to the spine, and \(T_R\) is the period of rest between any two work periods (\(T_W\)).

Equation (1) describing the NIEMG behavior during each of the work periods is rewritten as eq. (3):

\[
\text{NIEMG}(t) = A e^{-t(T_W + T_R)/T_{ss}}\frac{[t - n(T_W + T_R)]}{T_{ss}^2} + \text{NIEMG}_{ss}
\]

\[
(n + 1)\frac{T_W + T_R}{n(T_W + T_R)} + \text{NIEMG}_{ss}
\]

It was assumed that A and NIEMG_{ss} are not constant throughout the work/rest periods and are changing from one work period to the next. Furthermore, it was assumed that \(T_1\) might not be the same for all work periods.

Because this study used rest for only 10 minutes, the first transient component of eq. (2) will be dominant and the steady-state component contribution as well as the delayed hyperexcitability term can be neglected for this particular case. During the rest periods, therefore, the modified eq. (4) is:

\[
\text{NIEMG}(t) = (t - [(n - 1)T_W + nT_R])
\times \frac{B e^{-t(T_W + T_R)/T_{ss}}\frac{[t - [(n + 1)T_W + T_R]]}{T_{ss}^2} + \text{NIEMG}_{ss}}{(n + 1)(T_W + T_R)}
\]

It was also assumed that the amplitudes of NIEMG_{ss} and B would vary from one rest period to the next and that \(T_2\) may vary as well.

Similarly, the equation describing the development of displacement, a reflection of creep of the viscoelastic tissue, during a series of work periods separated by rest periods is given by eq. (5):

\[
\text{DISP}(t) = \frac{D_0 + D_L(1 - e^{-\frac{t - n(T_W + T_R)}{T_5}})}{n(T_W + T_R)}
\]

in which \(\text{DISP}(t)\) is the displacement as a function of time; \(D_0\) is the elastic component of amplitude; \(D_L\) is the viscoelastic component of amplitude; and \(T_5\) is the time-constant governing the development of creep during flexion.

The recovery of the displacement during the rest periods is described by eq. (6):

\[
\text{DISP}(t) = \frac{D_0 + D_L + (D_L - D_0) e^{-\frac{t - n(T_W + nT_R)}{T_5}}}{(n + 1)(T_W + nT_R)}
\]

\[
(n + 1)\frac{T_W + T_R}{n(T_W + nT_R)}
\]

such that R is the residual creep at the end of each rest period and \(T_5\) is the time-constant governing
Again, $D_0$, $D_1$, and $R$ were assumed to be a variable from one work/rest session to the next. $T_{n0}$ and $T_{n1}$ were also assumed to vary from one session to the next.

The long-term recovery after the work/rest session is modeled by eq. (2). Once the mean ± SD values of the experimental data were calculated, attempts were made to generate the best-fit models just described using the Marquardt–Levenberg nonlinear regression algorithm; in some cases, the algorithm failed to converge satisfactorily, and the initial or final values or both were arrived at by sequential recursive iteration, optimizing for regression coefficient.

A two-way analysis of variance (ANOVA) was performed to assess the effect of time after loading and repetitions on the recovery of the NIEMG and the displacement of the three protocols adopted on all the NIEMG and displacement at the three lumbar levels examined (L3–4, L4–5, L5–6). A Fisher post hoc test was applied, and the level of significance was set to $P < 0.05$ in order to confirm significant differences.

RESULTS

A typical recording from a preparation subjected to three sessions of load/rest followed by a 7-hour recovery period is shown in Figure 1. Randomly appearing, large-amplitude spasms (unpredictable EMG discharge) are superimposed on the generally decreasing EMG response over time. In addition, the strong spasms occurred at the end of the first 10-min session, causing the stiffness of the spine to increase and thereby reducing sharply the displacement at that point.

In general, the mean displacement, shown in Figures 2, 3, and 4, increased slowly over the first session and partially recovered during the first rest period. A similar pattern of increased displacement during loading and partial recovery was seen in the subsequent load–rest periods, with the final displacement at the end of the last loading period reaching the maximal observed. During the 7-hour recovery period, the displacement gradually decreased toward the initial displacement observed in the first loading period. The initial displacement value, however, was not reached by any preparation at the end of the 7-hour rest, indicating that a longer period of rest was required for complete recovery. The specific values of initial and final displacements as well as the associated creep are given in Table 1.

The general mean NIEMG pattern demonstrated a large-amplitude discharge at the beginning of the first loading session, with a gradual decrease throughout the session. The rest periods allowed partial recovery of the mean NIEMG, with a further
decrease throughout the following loading sessions, as shown in Figures 2, 3, and 4. After the first 10–20 minutes of the 7-hour recovery, a transient increase of the mean NIEMG was observed, followed by a small decrease and then a slow increase to the end of the 7-hour rest. In the groups subjected to six and nine repetitions, the final mean NIEMG at the end of the 7-hour recovery exceeded unity, indicating the presence of delayed hyperexcitability. The specific mean NIEMG values at the various stages of the experiment are given in Table 2.

The best-fit model is superimposed on the mean (±SD) of the experimental data shown in Figures 2, 3, and 4, respectively. The models developed are in good agreement with the experimental data, resulting in $R^2$ values near 0.9, except in a few cases resulting from the presence of random, unpredictable spasms interrupting the smooth changes in the EMG discharge during the work–rest periods. The models for the 7-hour recovery for the three experimental groups are superimposed on the data in Figure 5.

The statistical analysis revealed a significant effect of time after loading ($P < 0.0001$) and repetition ($P < 0.001$) for the L3/4, L4/5, and L5/6 NIEMG, confirming that the NIEMG was indeed changing over time and that the post-loading NIEMG responses of the three experimental groups were significantly different from each other. This implies that the group subjected to six repetitions demonstrated a delayed hyperexcitability, whereas the group exposed to three repetitions did not. Furthermore, the group subjected to nine repetitions had a delayed hyperexcitability of larger magnitude than the group subjected to six repetitions. The analysis also shows that an interaction of time and repetition was not present.

**DISCUSSION**

The major finding of this study was the effect of increasing the number of repetitions of cyclic loading on the development of a pronounced delayed hyperexcitability of the multifidus muscles. In particular, repeating a 10-minute session of cyclic lumbar loading three times does not elicit an acute neuromuscular disorder, whereas repeating the sessions six or nine times does so. Furthermore, the acute disorder seems to be of larger magnitude and have an earlier onset after the nine than the six repetitions. Because the delayed hyperexcitability reflects...
an acute inflammatory response, the viscoelastic tissues should be prevented from further exposure to strain in order to prevent increased damage that could lead to chronic inflammation and a chronic neuromuscular disorder over time.

The effect of repeating loading sessions was explored earlier for static lumbar loading and demonstrated that three or six repetitions of 10-minute loading separated by rest for 10-minute periods will not elicit a delayed hyperexcitability. From our present results, cyclic lumbar loading seems to have a more deleterious effect on the lumbar spine, as six repetitions are sufficient to elicit a pronounced and prolonged delayed hyperexcitability. As the acute inflammation and associated acute neuromuscular disorder reflect microdamage to the viscoelastic tissues, cyclic flexion-extension seems to cause more microdamage to the tissues than a single flexion-and-hold action (i.e., static flexion) for the same duration. Repeated stretch and release of viscoelastic tissues, therefore, may be a more intense stimulus than a steady, continuous stretch, leading possibly to more creep.

The cumulative creep percentages at the end of the three, six, and nine cyclic loadings were 85.1%, 115.9%, and 98.8%, respectively. Thus, creep indeed increased for higher numbers of repetitions, but not linearly. Similarly, the residual creep levels recorded at the end of 7-hour rest after cyclic loading were 39.3%, 31.7%, and 17.3% for the three-, six-, and nine-repetition groups, respectively. The residual creep was expected to be larger for higher repetitions of cyclic loading, but this was not the case. The six- and nine-repetition groups showed a pronounced delayed hyperexcitability at the terminal phase of the 7-hour recovery period. The 20%–70% increase in muscle activity during that period must increase the stiffness of the lumbar spine, which in turn masks the true residual creep in the viscoelastic tissues. The effect of increased stiffness, therefore, is to artificially decrease the residual creep. The group subjected to nine repetitions had the highest increase in magnitude (near 70%) of delayed hyperexcitability, highest lumbar stiffness, and lowest residual creep (17.3%). The group subjected to six repetitions had milder delayed muscular hyperexcitability (near 25% increase), but milder stiffness, result-

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<th><strong>Table 1. Displacement and creep.</strong></th>
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<td><strong>No. of repetitions</strong></td>
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<td>3</td>
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Initial, initial displacement at beginning of the first 10 minutes of loading; final, displacement value recorded at end of the 3-, 6-, or 9-repetition sessions; creep, percent elongation of ligament (soft tissues) at the end of repetition sessions; residual creep, amount of creep remaining in spinal soft tissues at the end of 7-hour rest.

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<th><strong>Table 2. Values of the normalized integrated EMG.</strong></th>
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<td><strong>No. of repetitions</strong></td>
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Initial, NIEMG recorded at beginning of first 10-min loading session (the base for normalization); final, NIEMG at end of last loading session; initial hyperexcitability, NIEMG after the first 10-min rest of the 7-h recovery; delayed hyperexcitability, NIEMG at the end of the 7-h rest.
ing in a residual creep of 31.73%; the three-repetition group did not show delayed hyperexcitability or increased lumbar stiffness, yielding the largest residual creep of 39.3%.

Similarly, the cumulative creep at the end of cyclic loading sessions could be masked by the spasms seen during that period. A typical example is the distinct spasms shown at the end of the first 10-minute session of Figure 1. The displacement shows a sharp decrease during that period, elucidating the effect of the spasms. In general, spasms seemed to be more frequent in later loading sessions when the cumulative microdamage in the tissues had increased. This, in turn, may have stiffened the spine and reduced the cumulative creep.

Overall, due to the masking effect of spasms and delayed muscular hyperexcitability, the creep recordings are probably not a reliable measure of the true laxity and microdamage in the viscoelastic tissues during that period. This, in turn, prevents a reliable relationship to be established between the magnitude of creep, microdamage, and delayed neuromuscular disorder.

The acute neuromuscular dysfunction present consisted of a decreasing reflex EMG response with random spasms during the loading sessions and a transient hyperexcitability followed by a delayed hyperexcitability during the 7-hour recovery. The differences among the three groups are highlighted in Figure 5. The NIEMG decreased throughout all working sessions for each group, with the final NIEMG of the nine repetitions being the lowest value for all lumbar regions. The first 10-minute period of recovery was marked by a sharp increase in the EMG response, which subsequently decreased for all groups. The initial hyperexcitability has been attributed to a reflex response of the muscles to prevent or limit further damage to the viscoelastic structures by stiffening of the joints.7,13,20,23,25,28,31

After the first hour of recovery, the six- and nine-repetition groups displayed a slow but distinct increase in mean NIEMG response, whereas the mean NIEMG from the three-repetition group gradually increased toward its initial level. The mean NIEMG, indeed, exceeded the preload value of unity within the first 3 hours of recovery for the six-repetition

**FIGURE 5.** The mean NIEMG of the three groups during the 7-hour recovery period are superimposed, highlighting the delayed hyperexcitability in the groups subjected to six and nine repetitions. (●) $3 \times 10^{-12}$; (■) $6 \times 10^{-10}$; (▲) $9 \times 10^{-10}$. Two-way ANOVA indicated a significant effect of repetitions and the post hoc test showed a significant difference between each of the curves.
group and within 2 hours for the nine-repetition group. Furthermore, after 7 hours of recovery, the mean NIEMG was above the preload value of unity in the six- and nine-repetition groups at all lumbar regions. In the latter group, the NIEMG magnitude at that time was significantly higher than in the six-repetition group. Overall, the mean NIEMG in the three-repetition group had risen to a value approaching the preload value, but not above it, suggesting that acute inflammation had resolved. Conversely, in the high-repetition group, the faster, earlier, more pronounced, and longer-lasting hyperexcitability suggests that an acute inflammation developed and that increasing the number of repetitions is a risk factor for a neuromuscular disorder.

The normal physiological reflex muscular response activated via fast- and slow-adapting mechanoreceptors in the viscoelastic tissues is overridden by the onset of an acute neuromuscular disorder that triggers a sustained hyperexcitability, stiffening the spine and protecting the damaged viscoelastic tissues. The tension usually generated by spinal ligaments, for example, is replaced by active tension from the muscles. The presence of muscular hyperexcitability in humans with low-back disorders has been confirmed repeatedly by electromyography and it is known to also decrease the normal range of motion of the lumbar spine.

Statistical analysis further confirmed the data and the models, indicating that the mean NIEMG curves during recovery were significantly different for the three experimental groups. This verified the hyperexcitability in the six- and nine-repetition groups as well as the higher intensity of the disorder in the nine-repetition group.

In conclusion, the present results provide physiological and biomechanical confirmation of the epidemiological prediction that a high number of repetitions of cyclic lumbar flexion leads to the development of an acute neuromuscular disorder. Increasing the number of repetitions demonstrated profound effects on the viscoelastic tissue and a breaking point appeared to be present between three and six repetitions of lumbar flexion/rest sessions. A rest period of 7 hours was not sufficient for recovery of the creep and associated microdamage induced in the ligamentous tissue. Notably, higher numbers of repetitions resulted in a disorder of higher magnitude. These data may provide valuable guidance in devising work schedules that avoid or prevent work-related musculoskeletal disorders and sports-related injuries. Early evidence is already available to confirm that many of the physiological and biomechanical processes observed in the cat are also observed in humans.

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