Available online at www.sciencedirect.com
SCIENCE DIRECT®
Clinical Biomechanics 18 (2003) 890–898
CLINICAL BIOMECHANICS
www.elsevier.com/locate/clinbiomech

Biomechanics and electromyography of a cumulative lumbar disorder: response to static flexion

M. Solomonow *, B.-H. Zhou, R.V. Baratta, E. Burger

Occupational Medicine Research Center, Bioengineering Laboratory, Department of Orthopaedic Surgery, Louisiana State University Health Sciences Center, 2025 Gravier Street—Suite 400, New Orleans, LA 70112, USA

Received 11 April 2003; accepted 1 July 2003

Abstract

Objective. To assess the mechanical and neurological processes active in the development of a cumulative trauma disorder (CTD) associated with repetitive exposure to periods of static lumbar flexion.

Methods. The spine of the feline model was subjected to a series of three 10 min sessions of static lumbar flexion with each session followed by a 10 min rest. A 7 h rest period was implemented after the series of three flexion-rest sessions while monitoring viscoelastic (disks, ligaments, etc.) creep and multifidus EMG. A model was fitted to the experimental data from the flexion-rest period and the 7 h recovery period.

Results. The creep developed in each 10 min static flexion period did not fully recovery during the following 10 min rest, resulting in a large cumulative creep at the end of the flexion-rest period. The cumulative creep did not fully recover over the following 7 h rest period. A neuromuscular disorder consisting of reduced muscular activity superimposed by spasms during static flexion periods and hyperexcitability during the 7 h recovery was evident. Comparison of the data to previous tests of continuous static flexion for 20 min reveal that the neuromuscular disorder elicited by the series of three 10 min flexion-rest was substantially attenuated when compared to a single 20 min static flexion although the overall work time was 50% larger.

Conclusions. Frequent rest periods are highly beneficial in attenuating the development of a CTD, yet not able to prevent it, as viscoelastic tissues residual creep accumulates and its recovery is of extremely long duration.

Relevance

The data provides direct biomechanical and physiological evidence that explain the development of a CTD due to prolonged exposure to static lumbar flexion as well as confirms the epidemiological data correlating such work conditions with substantial increase in symptoms of low back disorders. The benefit of frequent rest periods in attenuating the risk of such a disorder is validated as an effective intervention.

1. Introduction

The epidemiological literature identify occupational activities requiring periods of static joint flexion as a risk factor for the development of musculoskeletal disorders (National Academy of Sciences, 2001; Silverstein et al., 1986; Punnett et al., 1991; Hoogendoorn et al., 2000). In particular, the cumulative effect of performing such static activities over hours, days and years is recognized as cumulative trauma disorders (CTDs) (National Academy of Sciences, 2001; McGill, 1997; Marras, 2000). CTD, therefore, is the result of a three component dose-duration process consisting of the magnitude of the load sustained/applied, the duration over which the load is sustained/applied and the frequency at which such periods are repeated over a day (Silverstein et al., 1986). Evidently, lighter loads, shorter application duration and low repetition rates may have positive effects in retarding the development of CTD (Silverstein et al., 1986; National Academy of Sciences, 2001).

While CTD is defined epidemiologically (i.e., relating work activity to symptoms), the physiological and biomechanical processes (i.e., the pathology) active in its development are poorly understood and mostly

* Corresponding author.
E-mail address: msolom@lsuhsc.edu (M. Solomonow).

0268-0033/$ - see front matter © 2003 Elsevier Ltd. All rights reserved.
doi:10.1016/S0268-0033(03)00173-6

unexplored. It is thought (National Academy of Sciences, 2001) that submaximal strain developed in the viscoelastic tissues (ligaments, capsules and disc in the spine) during a work period does not fully recover before the next work period, giving rise to residual strain which accumulates during the work day. Furthermore, residual strain from a previous work day is compounded by the strain of the current work day, and so on for weeks, months and years. At some point, the residual strain becomes chronic, resulting in a permanent disability. This theory is based on a large number of epidemiological studies related to various occupations which require the use of the extremities and the spine (National Academy of Sciences, 2001). Full scientific (i.e., experimental) validation of the above hypothesis is still lacking. Our efforts to determine the development of a low back disorder in the in vivo feline model subjected to static or cyclic flexion gave some insights to this issue (Solomonow et al., 2003a, Claude et al., 2003; Williams et al., 2000; Jackson et al., 2001). To date we determined that static or cyclic lumbar flexion performed over a period of 20 min develops creep in the spinal viscoelastic tissues, as well as elicits spasms in the multifidus muscles. The experimental data further shows hyperexcitability of the muscles lasting well past 7 h of rest. Preliminary studies also show that acute inflammation developed in the ligaments 2–3 h after the long rest period started (Solomonow et al., 2003b). The creep developed in the viscoelastic tissues and the muscular hyperexcitability were predicted by our model to fully recover only after 24–48 h of rest. Overall, the new insights show that creep developing in the viscoelastic tissues during static flexion is not an isolated mechanical phenomena as it is also associated with a transient neuromuscular disorder and that recovery of the viscoelastic creep and the muscular disorder is a long process, outlasting the period over which it developed by more than 60 times. Consequently, the length and frequency of the rest periods will have to be included in any dose-duration formula assessing the risk of a given occupational activity.

We hypothesize that a series of short static lumbar flexion periods spaced by equally long rest periods will develop creep in the viscoelastic tissues that will not fully recover over the rest periods, giving rise to cumulative residual creep at the end of the work/rest session. We further anticipate that a 7 h recovery period in complete rest will not be sufficient for full recovery of the cumulative creep developed in the spine, leaving a residual creep for the "next work day". Based on our previous experience, we also expect that muscle spasms will be associated with the development of creep along with marked changes in the pattern of reflex activity from the multifidus muscles for many hours into the recovery period. Biomechanical/physiological verification of the hypothesis can confirm part of the theoretical infrastructure of CTD as related to the lumbar spine and may add new insights which may help to prevent or attenuate the disorder.

2. Methods

Preparation. Seven adult cats weighing 4.11 (SD: 0.37) kg were anesthetized with a single intraperitoneal injection of chloralose (60 mg/kg) in a protocol approved by the Institutional Animal Care and Use Committee (IACUC). The skin over the lumbar spine was dissected from the thoracic level to the sacral level and allowed to retract laterally to expose the dorsolumbar fascia. After dissection, the preparation was placed in a rigid stainless steel frame that allowed the isolation of the lumbar spine by external fixation (discussed in the next section). A gauze pad soaked with saline was applied over the incision during the experiment to prevent the exposed tissue from drying.

Instrumentation. Three pairs of stainless steel fine wire electromyography (EMG) electrodes were inserted, via hypodermic needles, into the multifidus muscles of the L-3/4, L-4/5 and L-5/6, on the right side, 5–6 mm from the midline. The wire electrodes were insulated except for a 1 mm exposed tip and the interelectrode distance of each pair was 3–4 mm. A ground electrode was inserted in the gluteus muscle. Each electrode pair constituted the input to a differential amplifier of 110 dB common mode rejection ratio, a gain capability of up to 200,000, and a band pass filter of 5–500 Hz. EMG response from each channel was monitored on oscilloscopes and stored in a computer at a sampling rate of 1000 Hz.

An "S" shaped stainless steel hook was inserted around the middle part of the L-4/5 supraspinous ligament and connected to the vertical actuator of a Bionix 858 Material Testing System (MTS, Inc., Minneapolis, MN, USA). The load was applied by the MTS actuator with a computer controlled loading system operated in load control mode. The vertical displacement of the actuator and the load cell output incorporated in it were also sampled into the computer along with the EMG data.

Two external fixators were used to isolate the lumbar spine; a first fixator to the L-1 posterior spinal process and a second fixator to the L-7 process. The external fixation was intended to limit the elicited flexion to the lumbar spine and to prevent interaction of thoracic and sacral/pelvic structures. The intention of the external fixation was not, however, to prevent any motion.

Protocol. The stainless steel hook applied to the L-4/5 supraspinous ligament was pulled up by the MTS actuator from a resting position to create lumbar flexion with a constant load of 40 N (which was determined in previous work (Claude et al., 2003) to be in the middle of the physiological range) for a 10 min period.

Following, the load was fully removed, allowing 10 min in the resting position. Three such load/rest periods were applied for a total cumulative loading in the static flexion time of 30 min.

During the 7 h of rest, 8 s tests were performed to assess vertical displacement (and creep) and EMG recovery. Tests were applied after 10 min of rest, 30 min of rest, 60 min and each hour thereafter. Each 8 s test consisted of a 6 s linear increase in load to 40 N followed by 2 s constant load of 40 N. The 8 s tests were recorded in 12 s windows triggered by the computer at the appropriate time. The spine remained unloaded between the specified tests (see typical tests in Fig. 1). The slow linear rate of increase in load over 6 s was used to avoid inflicting damage to the ligaments as is known to occur with the exposure to a sudden or fast stretch (Panjabi and Courtney, 2001). Similarly, the load was increased linearly to 40 N in the initial 6 s of each of the three 10 min loading period.

Analysis. 1.5 s windows of electromyogram, static load applied to the spine, and vertical displacement at the L-4/5 supraspinous ligament were sampled immediately at the beginning of the loading period, and every 20 s thereafter for each 10 min static loading periods, as well as for the short tests in the recovery period. Each electromyogram sample was full wave rectified and integrated over the 1.5 s window, and normalized with respect to the value obtained from the first window of the first 10 min period. The normalized integrated EMG (niEMG) of all the preparations subjected to the same load at the respective window was pooled, and the mean and standard deviation was calculated and plotted on a niEMG versus time plot for each of the preparations.

Displacements of the respective window of all preparations subjected to the load were pooled, and the mean (SD) was calculated and plotted as displacement versus time plot.

Model. The model considered is based on our previous work where continuous 20 min static load was followed by a 7 h recovery period (Solomonow et al., 2003a, Claude et al., 2003). The niEMG during the static loading period was described as follows:

$$\mathrm{niEMG}(t) = A\mathrm{e}^{-t/T_1} + \mathrm{niEMG}_0 \tag{1}$$

where niEMG_0 is the steady state amplitude; A, the amplitude of the exponential component; T_1, the time constant of the exponential component, t, the time.

Similarly, the niEMG during the long term recovery was modeled as:

$$\mathrm{niEMG}(t) = tB\mathrm{e}^{-t/T_2} + E(1-\mathrm{e}^{-t/T_3}) + C(t-T_\mathrm{d})\mathrm{e}^{-(t-T_\mathrm{d})/T_4} + \mathrm{niEMG}_0 \tag{2}$$

where B, C and E are the amplitudes of each of the three terms. $tB\mathrm{e}^{-t/T_2}$ represents the initial transient hyperexcitability, which decays completely within 1 h while reaching its peak in the first 10 min. $C(t-T_\mathrm{d})\mathrm{e}^{-(t-T_\mathrm{d})/T_4}$ represents a delayed hyperexcitability, this term is initiated after the second hour of rest, having no effect in the

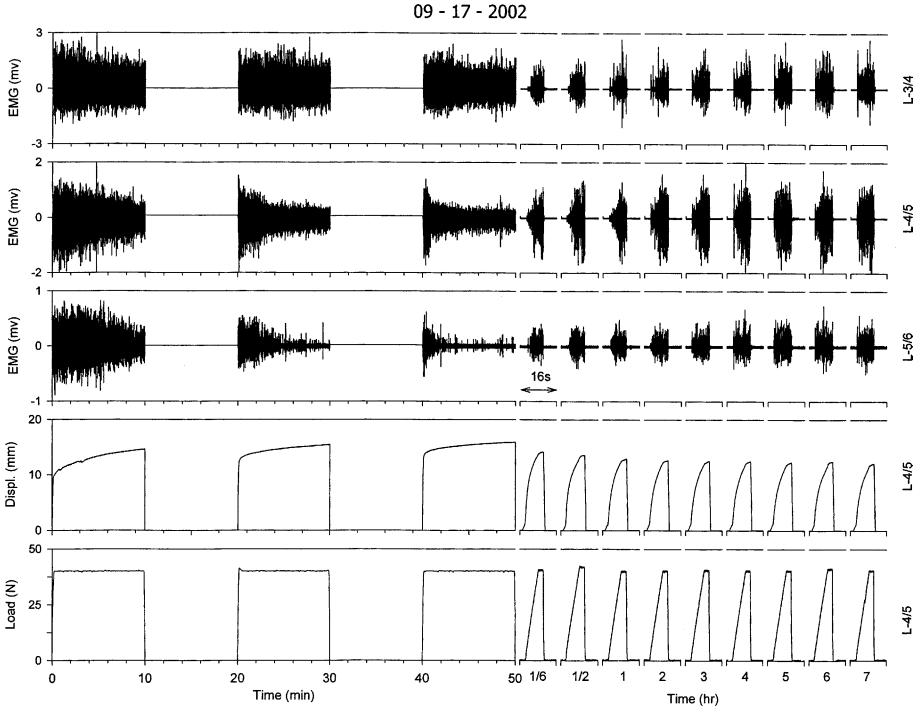

Fig. 1. A typical recording of load (bottom), displacement (and associated creep) and EMG from the multifidus muscles of the L-3/4 to L-5/6 lumbar levels during a three, 10 min load/10 min rest, session followed by 7 h of rest during which brief tests were conducted to assess recovery of creep and EMG.

first 2 h. $E(1-e^{-t/T_3})$ represents the steady state recovery, this term is a slowly rising exponential throughout the rest period. T_d is the time delay associated with the initiation of the delayed hyperexcitability. $niEMG_0$ 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 time components are defined:

T_w is the time period over which work (or load) was performed (or applied) by/to the spine.
T_R is the period of rest between any two work periods (T_w).

Eq. (1) describing the niEMG behavior during work period n is rewritten as:

$$\mathrm{niEMG}(t) = A_n \exp\left(\frac{-[t-n(T_w+T_R)]}{T_{n1}}\right)\Bigg|_{n(T_w+T_R)}^{(n+1)T_w+nT_R} + \mathrm{niEMG}_{0n} \quad (3)$$

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 may not be the same for all the work periods.

Since this study employs only 10 min rest, the first transient component of Eq. (2) will be dominant and the steady state component contribution as well as the delayed hyperexcitability term could be neglected for this particular case. During the rest periods, therefore, the modified equation is as follows:

$$\mathrm{niEMG}(t) = (t-[(n+1)T_w+nT_R])B_n \times \exp\left(\frac{t-[(n+1)T_w+nT_R)]}{T_{n2}}\right)\Bigg|_{(n+1)T_w+nT_R}^{(n+1)(T_w+T_R)} + \mathrm{niEMG}_{0n} \quad (4)$$

It was also assumed that the amplitudes of $niEMG_0$, and B will vary from one rest period to the next and that T_2 may vary as well.

Similarly, the equation describing the development of displacement (and indirectly creep in the viscoelastic tissues) during a series of work periods spaced by rest periods is given by:

$$\mathrm{Disp}(t) = \left[D_{0n} + D_{Ln} \times \left(1-\exp\left(\frac{-[t-n(T_w+T_R)]}{T_{n5}}\right)\right)\right]\Bigg|_{n(T_w+T_R)}^{(n+1)T_w+nT_R} \quad (5)$$

where $\mathrm{Disp}(t)$ is the displacement as a function of time, D_{0n} is the elastic component amplitude, D_{Ln} is the viscoelastic component amplitude, T_{n5} is the time constant governing the development of creep during flexion.

The recovery of the displacement during the rest periods is described by:

$$\mathrm{Disp}(t) = \left[D_{0n} + R_n + (D_{Ln} - R_n) \times \exp\left(-\frac{t-[(n+1)T_w+nT_R]}{T_{n6}}\right)\right]\Bigg|_{(n+1)T_w+nT_R}^{(n+1)(T_w+nT_R)} \quad (6)$$

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

Again, D_0, D_L and R were assumed to be variable from one work/rest session to the next. T_{n6} was also assumed to vary from one rest session to the next.

The long term recovery after the three work/rest sessions was modeled by Eq. (2).

Once the mean (SD) of the experimental data pooled from the seven preparations were calculated, attempts were made to generate the best fit models described above using the Marquardt–Levenberg non-linear regression algorithm; in some cases, the algorithm failed to converge satisfactorily; in these cases, initial and/or final values were arrived at by sequential recursive iteration, optimizing for coefficient of determinants (r^2).

3. Results

A typical recording of EMG from the L-3/4, L-4/5 and L-5/6, displacement and load from one preparation is shown in Fig. 1. In general, the EMG exhibited a gradual decrease during the periods of static flexion with superimposed spasms of unpredictable amplitude, duration or time of discharge. The EMG seems to partially recover its amplitude at the beginning of a following 10 min flexion (e.g., after 10 min rest), then continues to decrease further in the next work period. During the 7 h recovery period, a gradual increase of EMG over time was evident. The displacement trace exhibited the development of shear creep during the flexion periods with only partial recovery at the end of each rest periods. The residual creep at the end of each 10 min period of static flexion gradually increased from the first to the third flexion period. During recovery, a slow decrease in displacement over time was evident.

The mean (SD) of the niEMG and the mean (SD) of the displacement of the pooled data from all the preparations are presented in Fig. 2. The mean displacement increased from 12.2 mm in the beginning to 16.74 mm at the end of the first 10 min of static flexion corresponding to a shear creep of 27.13%. In the following 10 min of rest, a shear creep recovery of 9.8%, to a mean displacement of 15.1 mm took place. During the second 10 min of static flexion, the mean displacement increased again from 15.1 to 18.03 mm, representing a shear creep

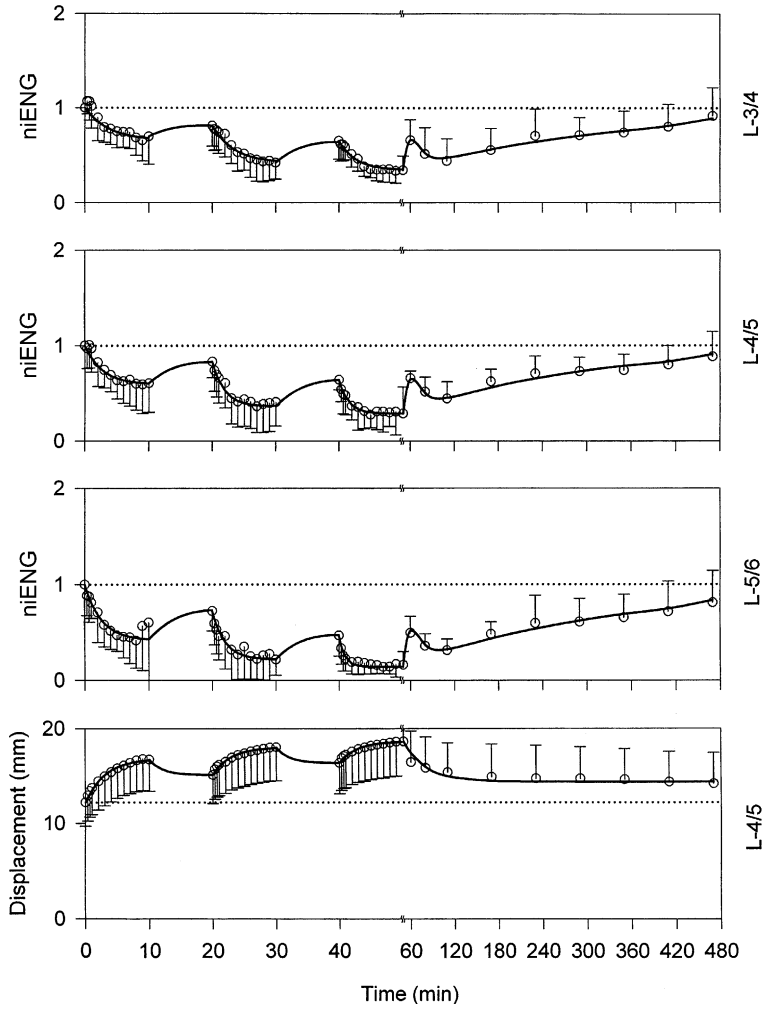

Fig. 2. The pooled mean and SD niEMG and displacement from the seven preparations during the loading and recovery periods. The models developed for the various segments of loading and rest is superimposed on the experimental data.

of 19.41%. The following 10 min rest allowed a creep recovery of 9.2%, to 16.37 mm. The final 10 min of static flexion resulted in an additional increase in the mean displacement to 18.64 mm, representing creep of 13.86%. Overall, the displacement increased during the three work/rest periods from 12.2 to 18.64 mm, or 52.77% cumulative shear creep.

During the 7 h of recovery, the mean displacement exhibited a gradual decline with a final value of 14.18 mm at the end of the 7th h. This corresponds to a 16.27% residual shear creep, i.e. the creep did not fully recover after 7 h of rest.

The reflex EMG from the multifidus muscles demonstrated a decreasing trend during static flexion and partial recovery during rest. During the first 10 min of static flexion, the mean niEMG from the L-3/4 to L-5/6 multifidi decreased from 1.0 to a range of 0.605–0.698% or 30–39.8% decrease. The following 10 min of rest allowed a recovery ranging from 11.3% to 22.3%. The second static flexion period caused additional decrease in mean niEMG ranging from 0.217 to 0.42. The second 10 min of rest allowed recovery to levels ranging from 0.651 to 0.471. The final static flexion period elicited further decrease in the mean niEMG to a range of 0.161–0.343. Overall, the cumulative decrease in mean niEMG over the three work/rest periods was 65.7%, 70.7% and 83.39% in the L-3/4, L-4/5 and L-5/6, respectively.

During the 7 h recovery period the mean niEMG exhibited two recovery waves. The initial wave of recovery, observed to peak within the first 10 min after static flexion, was terminated within the first hour. The mean niEMG increased from 0.343 to 0.658, from 0.293 to 0.661 and from 0.161 to 0.498 in the L-3/4, L-4/5 and L-5/6, respectively. The EMG decreased moderately in the following hour, after which a slow increase dominated the pattern of recovery throughout the remaining 6 h. The final values were 0.913, 0.883 and 0.81 for the L-3/4, L-4/5 and L-5/6, respectively. Full recovery was not achieved.

The best fit models derived from the pooled data of the seven preparations is superimposed on the experimental data shown in Fig. 2, and is summarized in Tables 1 and 2. A schematic representation of various parameters used in the model development in each work/rest session and in the recovery are shown in Fig. 3. The model describing the niEMG during the work periods confirms the assumptions that T_{n1} and niEMG_{0n} are indeed changing from one work session to another (see Table 1). Both demonstrated a gradual pattern of decrease from one work period to the next. For L-3/4, T_{n1} decreased from 3.7 to 3.4 and to 2.7 min and it decreased from 3.1 to 2.0 and to 1.5 min for the L-4/5 level. In the L-5/6 level, T_{n1} decreased from 2.6 to 2.0 and to 0.9 min.

Similarly, niEMG_{0n} decreased from 0.656 to 0.42 and to 0.343 in the model of the L-3/4 level. For the model of the L-4/5 level it decreased from 0.598 to 0.370 and to 0.293 over the three work sessions. In the L-5/6 level, it decreased from 0.418 in the first work session to 0.217 and to 0.141 in the second and third work sessions, respectively.

Table 1
niEMG model parameters

	n	A_n	T_{nl} (min)	niEMG_{0n}	r^2
Work period: $\text{niEMG}(t) = A_n e^{-[t-n(T_w+T_R)]/T_{nl}+\text{niEMG}_{0n}}$					
L-3/4	0	0.344	3.7	0.656	0.852
	1	0.391	3.4	0.420	0.957
	2	0.308	2.7	0.343	0.966
L-4/5	0	0.402	2.7	0.598	0.947
	1	0.463	1.9	0.370	0.958
	2	0.432	1.5	0.293	0.978
L-5/6	0	0.582	2.6	0.418	0.881
	1	0.507	2.0	0.217	0.915
	2	0.330	0.9	0.141	0.920

	n	niEMG_{0n}	B_n	T_n2
Rest period: $\text{niEMG}(t) = [t-((n+1)T_w+nT_R)]B_n e^{-[t-((n+1)T_w+nT_R)]/Tn2} + \text{niEMG}_{0n}$				
L-3/4	0	0.698	0.035	9
	1	0.420	0.06	10
L-4/5	0	0.610	0.06	10
	1	0.370	0.067	11
L-5/6	0	0.430	0.075	11
	1	0.220	0.07	10

Level	E	T_3 (min)	B	T_2 (min)	C	T_4 (min)	T_d (min)	niEMG_0	r^2
Recovery: $\text{niEMG}(t) = E(1-e^{-t/T3}) + tBe^{-t/T2} + C(t-Td)e^{-(t-Td)/T4} + \text{niEMG}_0$									
L3-4	0.667	300	0.08	10	0.001	250	350	0.343	0.975
L4-5	0.739	250	0.09	10	0.001	250	370	0.293	0.961
L5-6	0.839	300	0.09	10	0.001	250	370	0.161	0.974

Table 2
Displacement model parameters

n	D_{0n} (mm)	D_{Ln} (mm)	T_{n5} (min)	r^2
Work period: $\text{Disp}(t) = D_{0n} + D_{Ln}(1-e^{-[t-n(T_w+T_R)]/Tn5})$				
0	12.199	4.642	2.7	0.989
1	15.098	2.931	2.8	0.976
2	16.367	2.270	2.7	0.970

n	D_{0n} (mm)	D_{Ln} (mm)	R_n (mm)	T_{n6} (min)
Rest period: $\text{Disp}(t) = D_{0n} + R_n + (D_{Ln}-R_n)e^{-[t-((n+1)T_w+nT_R)]/T_n6}$				
0	12.199	4.642	2.899	2
1	15.098	2.931	1.269	2

D_0 (mm)	D_L (mm)	R (mm)	T_6 (min)	r^2
Recovery: $\text{Disp}(t) = D_0 + R + (D_L-R)e^{-t/T6}$				
12.199	6.438	2.185	30	0.897

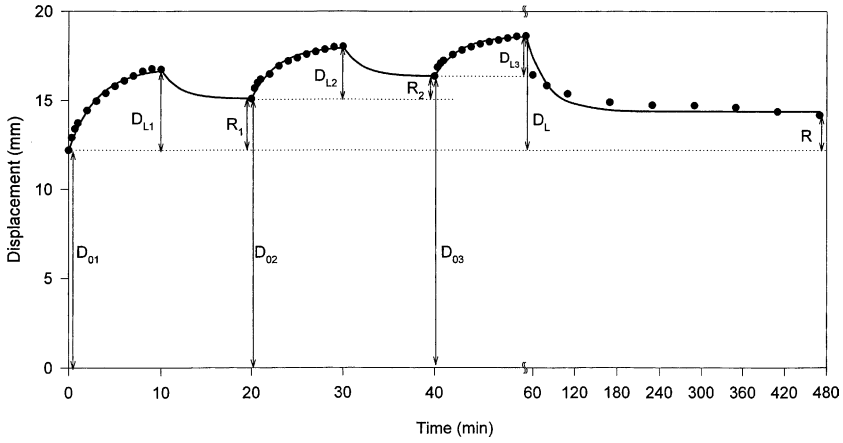

Fig. 3. Graphical depiction of the various terms and components of the models for the displacement and creep.

The exponential component amplitude A_n, however, was nearly constant for each lumbar level over the three work periods. For L-3/4 level it varied between 0.308 and 0.391 with no specific pattern of increase/decrease. Similarly, for L-4/5 it varied between 0.402 and 0.432. For L-5/6 the variations were between 0.507 to 0.582 and 0.303. Based on the relatively small variability and its randomness it is reasonable to assume that A does not change from one work period to the next. Overall, Eq. (3) could be simplified by replacing A_n by A and keeping it constant over the work periods. The best fit models to the data resulted in r^2 values ranging from 0.852 to 0.978 (see Table 1) indicating a very good fit.

The niEMG best fit model for the 10 min rest periods between the work periods indicates that T_{n2} is relatively constant, varying between 9 and 11 min. Similarly, B_n seemed to vary little between 0.06 and 0.075 with the exception of the first rest period of L-3/4 which was 0.035. niEMG_{0n}, however, was consistently decreasing in the second rest period for all levels, indicating a pattern.

The model for the long term recovery after the three work/rest sessions is given in the lower part of Table 1 demonstrating that T_2, T_3, T_4 and T_d are nearly identical for all lumbar levels tested. Similarly, the constant E, B and C were very similar across the three lumbar levels. Of importance, T_d varied between 350 and 320 min which corresponds to about 6 h delay in the initiation of the delayed hyperexcitability.

The best fit model for the displacement is also superimposed on the experimental data of Fig. 2 and is summarized in Table 2. The table points out that the time constant T_{n5} which governs the development of creep varied very little, between 2.7 and 2.8 min, and therefore could be accepted as constant over a series of work periods. D_{0n} and D_{Ln} however, demonstrated a distinct increasing and decreasing pattern, respectively, over the three work sessions. D_{0n} increased from 12.199 to 15.1 and to 16.367 over the three work periods whereas D_{Ln} decreased from 4.642 to 2.931 and to 2.27 over the same periods. The r^2 associated with the best fit models varied from 0.97 to 0.985 (see Table 2) indicating an excellent fit.

The best fit for the model during the rest periods shows that T_{n6} was 2 min in the two rest periods, yet D_{0n} was increasing with consecutive rest periods whereas D_{Ln} and R_n were decreasing with consecutive rest periods.

The model for the long term recovery is very similar to that developed previously, pointing out a T_{n6} of 30 min and residual creep R of 2.185 mm.

4. Discussion

The primary findings of this study consist of the fact that creep developed in the viscoelastic tissues of the spine during a period of static flexion under a moderate physiological load did not fully recover during an equally long rest period and accumulated over a series of such flexion/rest sessions to an astounding value of 52.77%. Seven hours of rest were not sufficient to allow full recovery of the creep developed during 30 min cumulative work, leaving residual creep for the "next work day". Muscles exhibited spasms and decreasing activity during the periods of static flexion, partial recovery during rest periods and a three wave recovery over the 7 h rest period, indicating a transient neuromuscular disorder. Overall, the data presented provide preliminary biomechanical and physiological validation to the theoretical understanding of CTD. It further adds to the current understanding by pointing out that a neuromuscular disorder is an integral part of such CTD.

Our previous work (Solomonow et al., 2003a, Claude et al., 2003) demonstrated that the shear creep developed in the lumbar spine during 20 min of static flexion did not fully recover after 7 h of continuous rest. Based on that, it was anticipated that a work/rest session of equal duration may present a more favorable condition

but will still not allow full recovery of the creep and therefore present a residual creep at the start of the next work period. However, it was not known if the time constant and magnitude governing the rate at which creep develops during flexion varies over consecutive periods of flexion. The data and the model developed point out that the time constant T_{n5} (about 2.7 min) does not vary over consecutive work periods. The amplitude of the exponential describing the development of creep. i.e., D_{Ln} in Eq. (5), however, decreases over consecutive work periods. This results in gradually decreasing creep in each work period. Overall, the development of creep at a given constant load static flexion over a long work/rest session will converge. In other words, the development of creep is not unlimited in a long work/rest session. It is still astounding to see that only three equally long work/rest sessions, as used in this study, resulted in 52.77% cumulative creep. An important point is the fact that the work/rest ratio was 1:1, which is a very liberal work schedule. Evidently, work requiring static flexion even at moderate load and equally long rest periods is not to be taken lightly as it imposes severe physiological changes with long term consequences.

The displacement behavior during the rest periods between the work sessions points out that T_{n6} is not changing from one work/rest session to the next, being 2 min. The magnitudes D_{0n} and D_{Ln} are increasing and decreasing, respectively, yet the residual displacement, R_n decreases. This reconfirms the previous observation that the creep developed in a long work/rest activity is limited and converges to a certain value. The recovery is governed, during rest, by a fixed time constant which under the conditions of this study allows reduction of creep with rest at the same rate.

The long term recovery of the displacement after the three work/rest sessions was relatively uneventful as it followed the anticipated pattern observed in our previous work (Solomonow et al., 2003a; Claude et al., 2003). The majority of the recovery occurred within the first hour at a time constant of $T_6 = 30$ min and the remainder was a slow asymptotic pattern leaving a residual displacement of 2.185 mm at the end of a 7 h rest. This implies that a 7–8 h overnight rest does not allow full recovery of the creep in the lumbar viscoelastic tissues, presenting some residual creep at the start of the next work day. It should be noted that the residual displacement obtained, 2.185 mm which corresponds to 17.9% creep, resulted only from 30 min of cumulative work. The residual may be significantly larger after a full days work.

The long term recovery of the niEMG after the three work/rest sessions bring out several important observations on the advantages of shorter work/rest schedule in comparison to a single longer work session.

The time delay T_d indicating the initiation of the delayed hyperexcitability ranged from 0 to 147 min (or <2.5 h) for a similar load after a straight 20 min of static flexion in our previous study (Solomonow et al., 2003a). The effect of 30 min cumulative work consisting of three, 10 min periods spaced by 10 min rest, significantly attenuated and postponed the delayed hyperexcitability. T_d was 350–370 min (or 6 h), indicating that it started much later. Furthermore, C, the amplitude of the delayed hyperexcitability was mostly 0.001 in the work/rest schedule (see Table 1) as opposed to 0.003 (three times larger) in the straight 20 min work studied previously. Overall, although the cumulative work time in the work/rest schedule was 50% larger in this study, the interposed rest periods significantly diminished the delayed component of the neuromuscular disorder associated with static flexion, and most likely attenuated the associated inflammation in the ligaments (Solomonow et al., 2003b). This point further stress out the value of appropriately spaced rest periods during work.

The initial hyperexcitability observed immediately as the 7 h recovery begun was also different in comparison to the straight 20 min static flexion. The amplitude, B, was about 0.08–0.09 for the three 10 min static load spaced by 10 min rest whereas it ranged between 0.13 and 0.3 when straight 20 min load and no rest were applied, indicating a less pronounced hyperexcitability. Similarly, the time constant governing the initial hyperexcitability, T_2 was 10 min compared to 11–12 min in the 20 min continuous load condition. This indicates a shorter and less intense initial hyperexcitability as a direct result of a work/rest schedule in comparison to a continuous loading without rest. Again, this should be taken while noting that the cumulative loading time was 30 min (50% longer) in this study relative to the 20 min of the previous one.

The time constant, T_3 and the amplitude, E associated with the steady state recovery of the niEMG were 250–300 min and 0.667–0.839, respectively. That was not much different from the respective data from the 20 min continuous static flexion being at 240 min and 0.5–0.7 (Solomonow et al., 2003a).

Overall, considering the different factors of the data and the associated model, it becomes clear that the rest periods inserted between the work periods resulted in substantially attenuated neuromuscular disorder associated with static flexion, but not in a complete prevention of such a disorder. Furthermore, the creep developed, and especially the residual creep carried over from day-to-day is important to note. The preliminary experimental data point out that residual creep is associated with acute inflammation of the ligaments (Solomonow et al., 2003b). If daily work continues to be imposed on the lumbar spine for prolonged periods, weeks or months, the inflammation can become chronic and lead to a permanent disability, as was found in clinical studies (Safran, 1985; Leadbetter, 1990). Based on the data evolving from this investigation and from

clinical experience, the chain of events leading to CTD elicited by static flexion consist of: daily accumulation of viscoelastic tissue creep, acute inflammation and transient neuromuscular disorder, residual creep carried over months/years, chronic inflammation and neuromuscular disorder, disability.

It should be emphasized that the data clearly points out the favorable effects of frequent and equally long rest periods. Yet, its effect was centered on attenuating the development of the neuromuscular disorder, not preventing it completely. It is still not clear nor obvious if there is a sufficiently long but realistic rest period that can prevent a CTD or how long can one work in jobs requiring periods of daily static flexion before a chronic disability occurs.

The data presented in this report was collected from the feline model: a quadriped, requiring justified extrapolation to human response. The creep developed in the viscoelastic tissues of the lumbar spine of humans during a period of static flexion was described by McGill and Brown (1992) as a first order response similar to the one developed in this report, confirming the similarity of the mechanical behavior of the viscoelastic tissues. Our recent report in which the spine of healthy humans was subjected to static flexion further confirms the creep as well as the neuromuscular disorder (Solomonow et al., 2003c). Spasms were present during the static flexion and significant changes in muscular activity, notably increased activity, were present after the static flexion. It is, therefore, possible to assert that the same disorder seen in the feline is present in humans, with the appropriate model constants re-calibrated for various differences.

5. Conclusions

The results of this investigation provided initial biomechanical/physiological validation to the theory of CTD. Furthermore, the results also point out that a neuromuscular disorder is an integral part of a CTD; spasms and muscular hyperexcitabilities are associated with creep in the lumbar viscoelastic tissues. The model developed can describe the development of creep and muscular activity during work, rest and following recovery and with some refinement may be used to develop work/rest schedules which may attenuate or prevent a CTD. The effect of frequency (or repetition rate) and load intensity are still missing from the model and require definition with additional experimental work.

The overall view of the scope of a CTD of this type seems much more severe than is currently perceived. The residual creep in the viscoelastic tissues also allows for excessive laxity in the intervertebral joints. Simultaneously, the viscoelastic tissues are inflamed and a neuromuscular disorder is present. The risk of developing a chronic laxity and inflammation is imminent and with it the potential of a permanent disability. Appropriate rest periods and limitation of a long term activity which promotes such a CTD may have significant effect on prevention of disability.

Acknowledgements

This work was supported by The National Institute of Occupational Safety and Health with Grants R01-OH-04079 and R01-OH-07622.

References

Claude, L., Solomonow, M., Zhou, B., Baratta, R.V., Zhu, M., 2003. Neuromuscular dysfunction elicited by cyclic lumbar flexion. Muscle Nerve 27, 348–358.

Hoogendoorn, W.E., Bongers, P.M., de Vet, H.C., Douwes, M., Koes, B.W., Miedema, M.C., Ariens, G.A., Bouter, L.M., 2000. Flexion and rotation of the trunk and lifting at work are risk factors for low back pain: results of a prospective cohort study. Spine 25, 3087–3092.

Jackson, M., Solomonow, M., Zhou, B., Baratta, R.V., Harris, M., 2001. Multifidus EMG and tension relaxation recovery after prolonged static lumbar flexion. Spine 26, 715–723.

Leadbetter, W., 1990. An introduction to sports induced soft tissue inflammation. In: Leadbetter, W., Buckwalter, J., Gordon, S. (Eds.), Sports Induced Inflammation. AAOS, Park-Ridge, IL.

Marras, W., 2000. Occupational low back disorder causation and control. Ergonomics 43, 880–902.

McGill, S., 1997. The biomechanics of low back injury: implications on current practice in industry and clinic. J. Biomech. 30, 465–475.

McGill, S., Brown, S., 1992. Creep response of the lumbar spine to prolonged full flexion. Clin. Biomech. 7, 43–46.

Musculoskeletal Disorders and the Workplace. National Academy Press, Washington, DC, 2001.

Panjabi, M., Courtney, T., 2001. High speed stretch of rabbit ACL: change in elastic failure and viscoelastic characteristics. Clin. Biomech. 16, 334–340.

Punnett, L., Fine, L., Keyserling, W., Herrin, G., Chaffin, D., 1991. Back disorders and non neutral trunk postures of automobile assembly workers. Scand. J. Work, Environ. Health 17, 337–346.

Safran, M., 1985. Elbow Injuries in Athletes. Clin. Orthop. 310, 257–277.

Silverstein, B., Fine, L., Armstrong, T., 1986. Hand and wrist cumulative trauma disorders in industry. Brit. J. Indust. Med. 43, 779–784.

Solomonow, M., Hatipkarasulu, S., Zhou, B., et al., 2003a. Biomechanics and electromyography of a common idiopathic low back disorder. Spine 28, 1235–1248.

Solomonow, M., Baratta, R.V., Zhou, B., Burger, E., Zieske, A., Gedalia, A., 2003b. Muscular dysfunction elicited by creep of lumbar viscoelastic tissues. J. EMG Kinesiol. 13, 381–396.

Solomonow, M., Baratta, R.V., Banks, A., Freudenberger, C., Zhou, B., 2003c. Flexion-relaxation response to static lumbar flexion in males and females. Clin. Biomech. 18, 273–279.

Williams, M., Solomonow, M., Zhou, B., Baratta, R., Harris, M., 2000. Multifidus spasms elicited by prolonged lumbar flexion. Spine 22, 2916–2924.