Available online at www.sciencedirect.com
ScienceDirect
Journal of Electromyography and Kinesiology 17 (2007) 142–152
JOURNAL OF ELECTROMYOGRAPHY AND KINESIOLOGY
www.elsevier.com/locate/jelekin

Work to rest durations ratios exceeding unity are a risk factor for low back disorder; a feline model

Paola Sbriccoli [a,b], Moshe Solomonow [a,*], Bing-He Zhou [a], Yun Lu [a]

[a] *Musculoskeletal Disorders Research Laboratory, Bioengineering Division, Department of Orthopaedic Surgery, University of Colorado at Denver and Health Sciences Center, RC-1 North Tower, Room 2103, 12800 East 19th Avenue, Denver, CO 80045, United States*
[b] *Department of Human Movement and Sport Sciences Exercise Physiology Laboratory, Istituto Universitario di Scienze Motorie (IUSM), Roma, Italy*

Received 6 October 2005; received in revised form 6 January 2006; accepted 30 January 2006

Abstract

Low back disorders are prominent among the work force engaged in static anterior flexion during the workday. As a continuing part of a long-term research aimed to identify the biomechanical and physiological processes and corresponding risk factors leading to such cumulative trauma disorder (CTD), we ventured to assess the effect of rest and the work-to-rest duration ratios that may prevent CTD. Three groups of the feline model were subjected to three load/rest paradigms: two 30 min loading periods spaced by 10 min rest in Group I, two 30 min loading period spaced by 30 min rest in Group II and one 60 min loading period for Group III. The cumulative loading duration in the three groups was 60 min. Each of the groups were allowed 7 h of rest while monitoring EMG and lumbar viscoelastic tissue creep each hour. The results demonstrate that for two 30 min load periods with a 30 min in between rest, an acute neuromuscular disorder was not present whereas for two 30 min loading with a 10 min rest it was. Similarly, for a 60 min loading with long-term rest, the disorder was present. Post hoc Fisher analysis demonstrated significant differences in the delayed hyperexcitability between the first and second group ($P < 0.0001$) and the third and second ($P < 0.0001$) group. Statistical difference in the displacement data of the three groups was not present. ANOVA showed a significant effect of time post-loading ($P < 0.0001$ and different rest durations ($P < 0.0001$) on the EMG data during the 7 h recovery. The new data allow us to conclude that a work-to-rest duration ratio of 1:1 can prevent the development of CTD as long as the work periods are not too long (<60 min). Longer static flexion durations do not respond favorably to rest even if it is of equal or longer duration. It is suggested that appropriate durations of rest may be a viable tool to avert CTD in a certain range whereas long static flexion durations should be avoided at all cost.

Keywords: Spine; Lumbar; EMG; Disorder; Muscle

1. Introduction

Static joint flexion performed by workers in the course of routine occupational activities was shown to lead to the development of a Cumulative Trauma Disorder (CTD) characterized by chronic joint pain, stiffness (spasms) of associated muscles, limited range of motion and weakness (National Academy of Sciences, 2001; Silverstein et al., 1986; Hoogendoorn et al., 2000; Punnett et al., 1991; Marras, 2000). The epidemiological literature cited above further determines that high load magnitude, high number of repetitions and high frequency of such activities are risk factors for the development of the disorder.

Our recent work focused on providing the missing physiological and biomechanical processes that lead to the development of CTD in the lumbar spine using a feline model subjected to static loads in anterior flexion. To date, we confirmed that high load magnitude (Sbriccoli et al., 2004a), high number of repetitions (Sbriccoli et al., 2004b), longer

* Corresponding author. Tel.: +1 303 724 0383; fax: +1 303 724 0394.
E-mail address: Moshe.Solomonow@UCHSC.edu (M. Solomonow).

1050-6411/$ - see front matter © 2006 Elsevier Ltd. All rights reserved.
doi:10.1016/j.jelekin.2006.01.014

loading durations (LaBry et al., 2004) and higher frequencies of flexion (Lu et al., 2004) are indeed risk factors. We also introduced the effect of a rest duration as a potential factor that may prevent/attenuate CTD or enhance it if longer or shorter durations were chosen, respectively (Courville et al., 2005). Rest periods of 10 min or more between each of six sequential sessions of 10 min of static lumbar loading of moderate magnitude were shown to have little impact towards the development of the disorder whereas rest periods of 5 min for the same loading sequence did contribute to the development of the disorder, (Courville et al., 2005). In general, the presence of the above risk factors (high loads, many repetitions, short rests, etc.) was manifested in a delayed but prolonged hyperexcitability of the posterior muscles that was related to the presence of creep and acute inflammation in the supraspinous ligaments due to microdamage generated in their collagen fibers (Solomonow, 2004; Solomonow et al., 2003b; Leadbeter, 1990; Woo et al., 1999). The general process that governs the development of CTD is considered as an acute inflammation of the collagen fibers which are further exposed to repeated periods of daily work until a chronic inflammation develops. CTD, therefore is the manifestation of a chronic inflammation. In our previous research, we considered any work, rest, repetition or load magnitude combinations that results in creep, spasms and delayed hyperexcitability resulting from acute inflammation as a risk factor (Solomonow et al., 2003b). This criteria is justified by the assumption that a worker who develops a disorder elicited by an acute inflammation due to a certain dose–duration of load and continues to be exposed to the same loading conditions in his daily work will, over time, develop a chronic inflammation and the associated CTD.

At the present, we can conclude that when the work and rest periods are of equal duration in a sequence of six, 10 min work/10 min rest paradigm at low and moderate load, CTD could be avoided. The conclusion above, however, may or may not be applicable if different work/rest durations are chosen. For example, 30 min work with 30 min rest versus 30 min work with 10 min rest. Another possibility revolves around the question if a ratio of work to rest of 1:1 can prevent CTD even if longer work and equally longer rest periods are chosen, such as 60 min of work followed by 60 min of rest sequence. The exploration of different lengths of work duration at a 1:1 ration with following rest are relevant from the ergonomic standpoints of work organization, scheduling and work rotation. For example, a worker who is allocated to a set of 10 min work with 10 min following rest for six times and totaling 2 h could be instead allowed 60 min of continuous work and then rotated to a different job activity that does not require static flexion. Such possibility can save 1 h and significantly increase productivity and efficiency. It is based, however, on the premise that 60 min of continuous work will not induce a disorder when equally long rest (or 1:1 work to rest ratio) is applied. Extending our knowledge on these issues, therefore, may allow generalization of the work to rest ratio as a factor in any dose–duration formula to be used to optimize work schedules.

The objectives of this study, therefore, are to determine if two 30 min static flexion periods with 10 and 30 min of intermediate rest can contribute to or prevent the development of CTD, respectively. Furthermore, we would like to determine if a work to rest ratio of 1:1 with 60 min work and 60 min rest will still prevent the initiation of a CTD. We hypothesize that two sequential periods of 30 min static load with 30 min intermediate rest will not lead to CTD. Conversely, if only 10 min of intermediate rest is allowed, early signs of CTD may develop in the lumbar viscoelastic tissues. Similarly, we also hypothesize that work to rest ratio of 1:1 with longer but equivalent work and rest periods will not prevent the initiation of CTD. The common factor in the different paradigms is the fact that the cumulative work period in any sequence is 60 min.

2. Methods

2.1. Preparation

Twenty adult cats with average weight of 2.93 ± 0.6 kg were used in this study. Cats were anesthetized with 60 mg/kg chloralose, according to a protocol approved by the Institutional Animal Care and Use Committee (IACUC). The skin overlying the lumbar spine was dissected to expose the lumbar fascia, and an S-shaped stainless-steel hook was applied around the supraspinous ligament between L-4 and L-5. The preparation was then positioned in a rigid stainless steel frame and fixed for subsequent EMG electrodes insertion. Preparations were divided into three experimental groups each subjected to different rest duration periods between following loading periods: two 30 min work and 10 min rest or $2 \times 30{:}10$ for the first group ($N = 7$), and two 30 min work and 30 min rest or $2 \times 30{:}30$ for the second group ($N = 7$). In the third group ($N = 6$), 60 min work followed by rest or $1 \times 60{:}60$ was used.

2.2. Instrumentation

The lumbar spine was isolated by means of two external fixators applied to the L-1 and L-7 posterior process, respectively. The external fixation was intended to limit the elicited flexion to the lumbar spine and to prevent interaction of thoracic and sacral/pelvic structures, but not to prevent any motion.

Three pairs of fine stainless steel wire EMG electrodes (inter-electrode distance: 3–4 mm) were inserted in the right L-3/4, L-4/5, L-5/6 multifidus muscles, 8-mm. laterally from the posterior spinal processes. A ground electrode was inserted into the gluteus muscle. Each electrode pair constituted the input to a differential EMG amplifier with a 110-db common mode rejection ratio, a gain of up to 200,000 and a band pass filter in the range of 6–500 Hz. The EMG was recorded with a sampling rate of 1000 Hz,

and it was continuously monitored on oscilloscopes. The S-shaped stainless-steel hook inserted around the L-4/5 supraspinous ligament was connected to the crosshead of the Bionix 858 Material Testing System (MTS, Inc., Minneapolis, MN, USA), in which a load cell was located. The load was applied through the MTS actuator with a computer controlled loading system in a load control mode. The vertical displacement of the actuator was also monitored continuously. The load cell and displacement outputs of the Bionix 858 MTS were sampled into the computer along with the EMG signals.

2.3. Protocol

The three experimental groups were subjected to the protocol described below. A pre-tension of 1 N was applied to the supraspinous ligament in order to standardize the initial conditions in all the different preparations (Eversull et al., 2001). For each experimental group, the same constant load (40 N) was applied to the lumbar spine via the S-shaped stainless-steel hook. The tension level was maintained constant during the loading (i.e., work) sessions in all groups. The loading periods were spaced by one 10-min rest periods (first group) and one 30-min rest period (second group) then followed by 7 h of rest. In the third group, a 60 min loading period was followed by a 7 h rest. Zero load was kept applied during rest periods. The EMG signal, the vertical displacement and the load were recorded continuously during the loading periods. Nine 8-s loading tests were performed during the following 7 h of recovery. This was obtained by a linear increase in tension over 6-s followed by 2-s of constant load. The linear increase in load over 6-s was used in order to avoid possible damage to the ligaments due to a sudden or fast stretch (Panjabi and Courtney, 2001). A 6-s ramp to the respective load of each experimental group was also applied in the initial loading of each 30 and 60 min working period.

The EMG, load and supraspinous ligament displacement data were then stored in the computer for subsequent analysis. For all the experimental groups, the load used was 40 N, and the cumulative working period was 60 min. The choice of 40 N was based on our previous work, being in the middle of the physiological range (Eversull et al., 2001) and not posing a risk factor (Sbriccoli et al., 2004a).

2.4. Data analysis

The analysis of the EMG, vertical displacement and the static load applied to the supraspinous ligament was performed over 1.5-s epochs. During the constant load periods the analysis was performed at the very beginning of the loading period (after the 6 s ramp) and then every 20 s for each static load period. During the recovery phase, the analysis was performed over the 2-s constant load phase following the 6-s ramp. In order to be confident that the load was reached, the first 0.5-s of the constant load phase (2-s duration) was discarded, and the analysis was performed over the following 1.5-s. Each EMG sample was integrated over the 1.5-s epoch and normalized with respect to the integrated EMG computed for the first window of the first constant load period to obtain the normalized integrated EMG (NIEMG). For each experimental group, all the corresponding NIEMG data were pooled together and the mean and standard deviation (SD) values were computed and plotted on a NIEMG versus time for each of the muscles of the three lumbar levels investigated. The displacement data were normalized to the displacement recorded at the beginning of the first loading period. Then, the corresponding normalized displacement data of each of the experimental groups (2×30:10, 2×30:30 and 1×60:60) were pooled together as mean ($\pm$SD) and plotted as normalized displacement versus time.

2.5. 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., 2002, 2003b,c). In order to convert the equations to describe a series of work periods spaced by rest periods, two new time components are defined: T_W is the time period over which work (or load) was performed (or applied) by/(to) the spine, which in this study corresponded to 30-min in the first and second group, and 60-min in the third group. T_R is the period of rest between the two work periods, T_W (10-min in the first group, 30-min in the second group and 60-min in the third group). The equation describing the NIEMG behavior during each of the work periods is rewritten as:

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

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

Since this study employs only short rest periods, the first transient component of the recovery equation will be dominant and the steady-state component contribution as well as the hyperexcitability term can be neglected (Solomonow et al., 2003b).

During the rest period, therefore, the equation is modified as follows:

$$\mathrm{NIEMG}(t) = (t-[(n+1)T_W+nT_R])B_n \mathrm{e}^{\frac{-\{t-[(n+1)T_W+nT_R]\}}{T_{n2}}}\Big|_{(n+1)T_W+nT_R}^{(n+1)(T_W+T_R)} + \mathrm{NIEMG}_{0n}$$

The equation describing the development of displacement (and indirectly creep in the viscoelastic tissues) during the work periods spaced by rest periods is given by

$$\mathrm{DISP}(t) = \left[D_{0n} + D_{\mathrm{Ln}} \left(1 - \mathrm{e}^{-\frac{[t-n(T_{\mathrm{W}}+T_{\mathrm{R}})]}{T_{n5}}} \right) \right] \Bigg|_{n(T_{\mathrm{W}}+T_{\mathrm{R}})}^{(n+1)T_{\mathrm{W}}+nT_{\mathrm{R}}}$$

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

$$\mathrm{DISP}(t) = \left[D_{0n} + R_n + (D_{\mathrm{Ln}} - R_n)\mathrm{e}^{-\frac{t-[(n+1)T_{\mathrm{W}}+nT_{\mathrm{R}}]}{T_{n6}}} \right] \Bigg|_{(n+1)T_{\mathrm{W}}+nT_{\mathrm{R}}}^{(n+1)(T_{\mathrm{W}}+T_{\mathrm{R}})}$$

The long-term 7 h recovery after the work–rest–work session was modeled by the original equation for long-term recovery (Claude et al., 2003; Solomonow et al., 2003b). Once the mean ± SD of the experimental data 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 or final values were determined by sequential recursive iteration, optimizing for regression coefficient.

A two-way analysis of variance (ANOVA) was performed to test the effect of load and time post-loading on the recovery of the L-3/4, L-4/5, L-5/6 NIEMG and the displacement data. Moreover, the effect of rest duration (10 and 30 min) on all the NIEMG and displacement at the three lumbar levels explored (L-3/4, L-4/5, L-5/6) was tested by the post hoc Fisher test. Significance was set at 0.05 for all statistical tests.

3. Results

Typical examples of EMG, load and displacement from two preparations, the first with two 30 min work sessions and 10 min rest and the second at 60 min work session followed by rest are shown in Figs. 1(a) and (b). In the first 30 min of loading, the EMG is progressively decreasing over time for all the three lumbar levels, the decrease being more evident during the second 30 min of loading. Note the presence of random spasms during the loading periods and during the 7 h of recovery. The recovery period is characterized by an initial increase in EMG followed by a decrease and than by a gradual increase to the end of the recovery period.

The mean (±SD) NIEMG and displacement data collected for the three experimental groups (2×30:10, 2×30:30, and 1×60:60) are shown in Figs. 2–4.

The mean initial displacement developed in the preparations subjected to the 2×30:10 protocol (Fig. 2) was 10.765 mm at the beginning of the first loading period, reaching a mean value of 17.92 mm (mean creep: 66.54%) at the end of the first 30-min load. During the 10 min of rest between loading periods the creep recovered to a mean value of 41.97%. The displacement further increased during the second loading period, up to a mean final value of 19.052 mm, corresponding to a mean creep of 76.98%. During the 7 h of recovery the displacement decreased to a mean final value of 11.642 mm, resulting in a mean residual creep of 8.14%.

For the group subjected to the 2×30:30 protocol (Fig. 3) the initial mean displacement was 11.022 mm. At the end of the first 30 min of loading, the mean displacement was 17.334 mm, corresponding to a mean creep of 57.36%. During the 30 min of rest between the two loading periods, creep partially recovered to a mean value of 30.58%. The second 30 min loading period resulted in a further increase in mean displacement to a final value of 18.127 mm, corresponding to a mean final creep of 64.46%. The 7 h of recovery were characterized by a progressive recovery of creep, resulting in a mean residual creep of 18.09% (mean displacement at the end of the 7 h of recovery: 13.016 mm).

For the group subjected to a single 60-min load (Fig. 4), the initial displacement was 8.980 mm. At the end of the 60-min period the displacement was 19.155 mm; corresponding to 113% creep. At the end of the 7 h rest the displacement was 9.887 mm, or a residual creep of 10.1%.

For the preparations subjected to the 2×30:10 protocol, the mean NIEMG showed a decrease during the first loading period, reaching values of 0.359, 0.309, and 0.317 for the L-3/4, L-4/5, and L-5/6, respectively. A partial recovery of the mean NIEMG was observed during the 10 min of rest between the two loading periods. The second loading period resulted in a further decrease in the NIEMG, and the mean values obtained at the end of the two loading periods were 0.307 (69.3%), 0.246 (75.4%), and 0.267 (73.3%) for the three lumbar levels considered. The 7 h of recovery consisted of an initial NIEMG peak to the values of 0.824, 0.876, and 0.917 in the first hour of recovery for the three lumbar levels considered. The NIEMG peak was followed by a gradual decrease during the following 2 h of recovery. Then the NIEMG gradually increased, exceeding the pre-load value (1.0) after the fourth hour of recovery for all the three lumbar levels. The mean final NIEMG values at the end of the 7 h recovery phase were 1.542, 1.375, and 1.643 at L-3/4, L 4/5, and L-5/6, respectively.

In the group subjected to the 2×30:30 protocol, the NIEMG decreased during the first 30 min loading period, reaching mean values of 0.2673 (73.27% decrease) for the L-3/4, 0.3987 (60.13% decrease) for the L-4/5, and 0.3152 (68.48% decrease) for the L-5/6. During the 30 min of rest between the two loading periods the mean NIEMG partially recovered to 0.7371, 0.9751, and 0.6878 in the L-3/4, L-4/5, and L-5/6, respectively. The second 30 min of loading resulted in a further NIEMG decrease. At the end of the two loading periods, the NIEMG reached the mean values of 0.21 (79% decrease) at the L-3/4, 0.3661 (63.39% decrease) at the L-4/5, and to 0.3121 (68.49% decrease) at the L-5/6. During the 7 h of recovery, the NIEMG showed an initial peak to mean values of 0.817, 0.977, and 0.858 for the L-3/4, L-4/5, and L-5/6, respectively. This peak was followed by a gradual recovery of the NIEMG throughout the recovery without reaching or

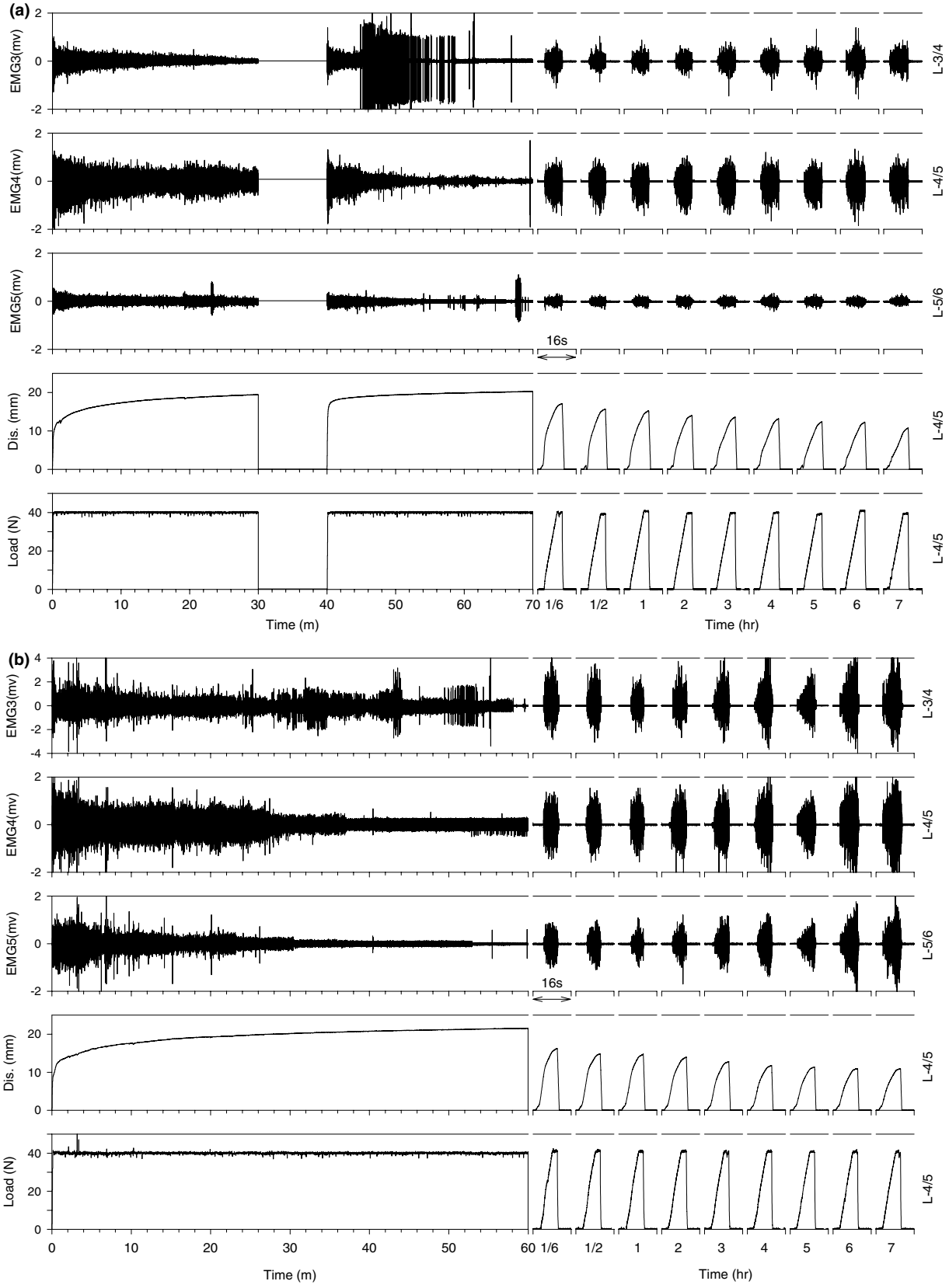


Fig. 1. (a) A Typical recording of EMG from the L-3/4, L-4/5, and L-5/6 multifidus (top three rows) and lumbar displacement and static load (bottom) recorded from one preparation subjected to the $2 \times 30{:}10$ protocol. (b) A similar recording from a preparation subjected to a 60 min of load with 7 h of rest.

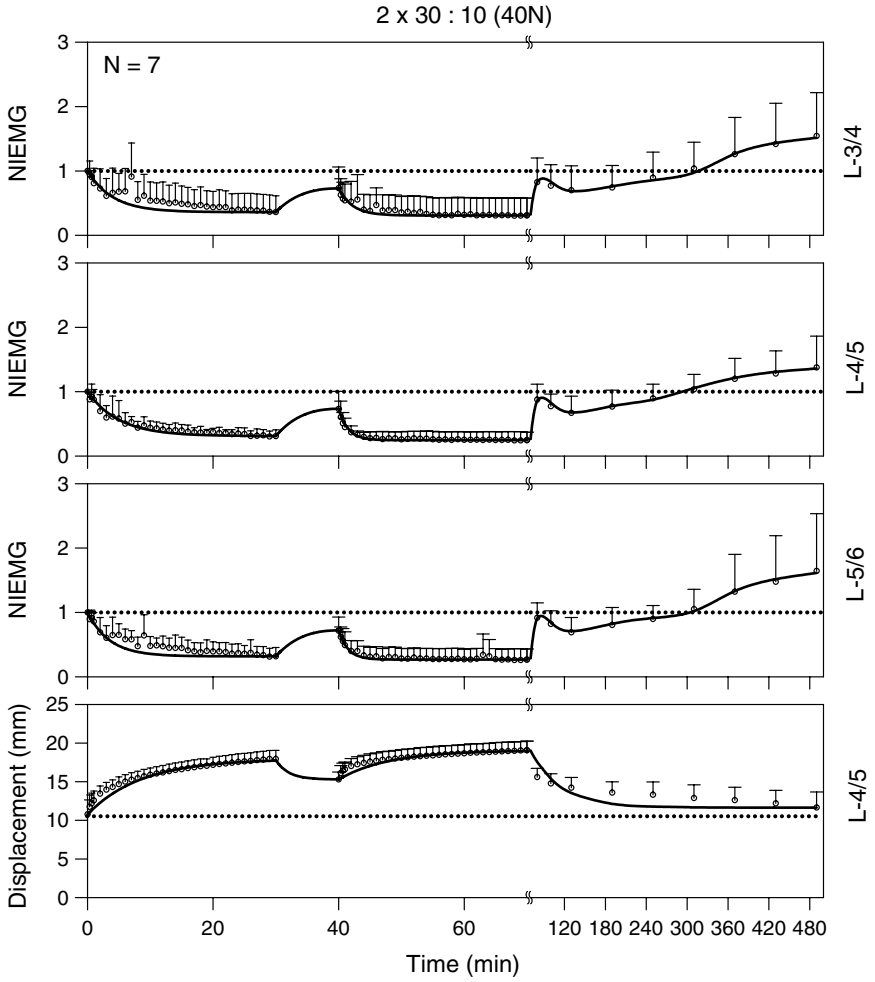


Fig. 2. The mean (±SD) NIEMG of the L-3/4, L-4/5, and L-5/6 multifidus as well as the mean displacement during the load–rest–load session and the 7-h recovery for the 2 × 30:10 group are shown. The solid lines through the data points represent the models developed to describe NIEMG and displacement during load–rest–load and long-term recovery.

exceeding the pre-load value of 1.0 at any of the three lumbar levels explored. At the end of the 7 h of recovery, the mean NIEMG was 0.678 for the L-3/4, 0.946 for the L-4/5, and 0.83 for the L-5/6.

In the third group, (1 × 60:60), the mean NIEMG decreased to 0.1795, 0.1963 and 0.2243 of its in initial value in the three lumbar values, respectively, at the end of the 60 min load. During the recovery period, the mean NIEMG peaked in the first hour to 1.3144, 1.3120 and 1.1252 in the three respective lumbar levels. It decreased slightly in the following hour, then increased, exceeding 1.0 in the third hour. The final mean NIEMG values were 1.7923, 1.7705, and 1.4015 at the end of the 7 h of recovery.

The best-fit model constants fitted to the NIEMG and displacement data were superimposed on the mean (±SD) of the experimental data presented in Figs. 2–4.

The R^2 values found for the models developed for NIEMG and Displacement were in good agreement with the experimental data, with values of about 0.9. Only in one case this value was 0.6, probably due to the presence of spasms within the EMG traces.

The differences observed between the 2 × 30:10 and the 2 × 30:30 protocols from Fig. 5 were confirmed by the statistical analysis. The post hoc Fisher test showed a significant difference ($p < 0.0001$) between the L-3/4, L-4/5, and L-5/6 NIEMG post-loading data obtained for the 2 × 30:10 protocol compared to those obtained in the 2 × 30:30 preparations. Significant differences were also present between the second (2 × 30:30) group and the third (1 × 60:60) group at ($p < 0.0001$). No difference was evident between the first (1 × 30:10) and third (1 × 60:60) Groups. The post hoc Fisher test failed to show any statistical differences as referred to the Displacement data among the three experimental designs. Finally, the ANOVA showed a significant effect of time post-loading ($p < 0.0001$) and different rest duration ($p < 0.0001$) on the NIEMG data during the 7 h of recovery for all the three lumbar levels explored (L-3/4, L-4/5 and L-5/6), thus indicating that the NIEMG changed significantly as time progressed, and that these changes were significantly affected by the duration of the rest period.

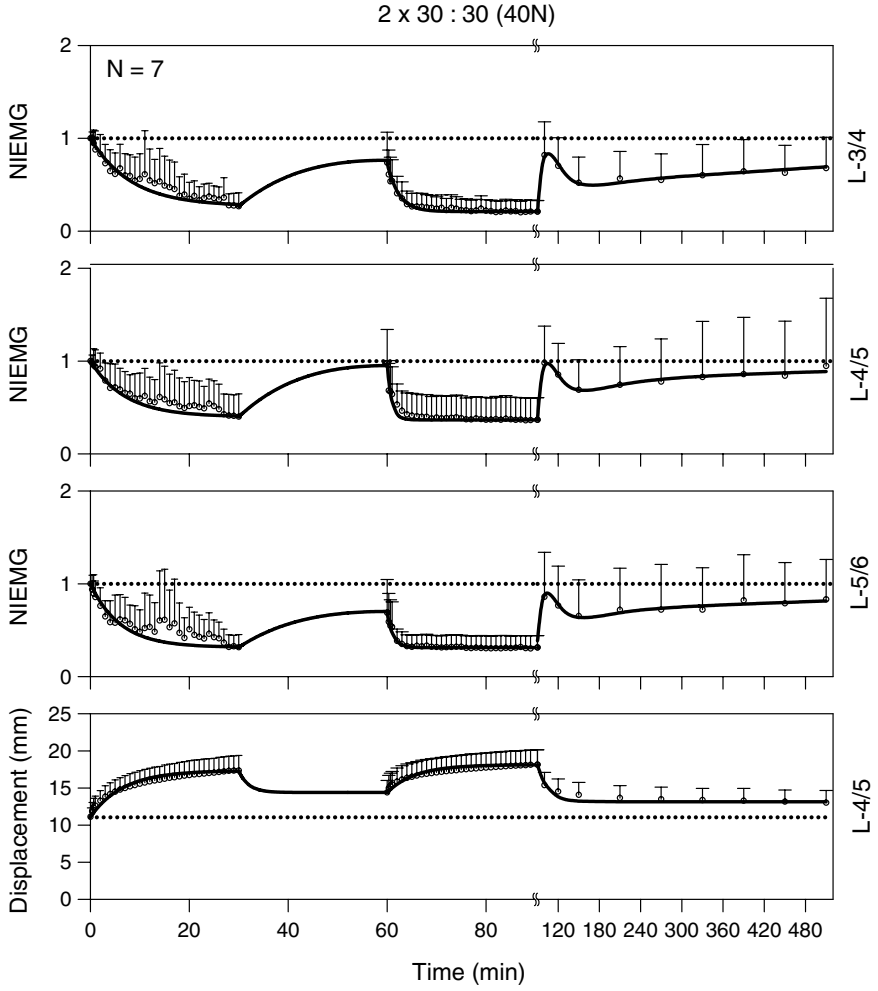


Fig. 3. The mean (±SD) NIEMG of the L-3/4, L-4/5, and L-5/6 multifidus as well as the mean displacement during the load–rest–load session and the 7-h recovery for the 2 × 30:30 group are shown. The solid lines through the data points represent the models developed to describe NIEMG and displacement during load–rest–load and long-term recovery.

4. Discussion

The major findings of this investigation, further confirm that a work/rest sequences of equal durations such as 10:10 min or 30:30 min, do not result in a delayed neuromuscular disorder that may lead to CTD over time. Sequences exceeding 1:1 ratio such as 10:5 min and 30:10 min, however, result in an acute neuromuscular disorder which constitutes the initial phase of a CTD. Furthermore, long work durations, near 60 min, elicit an acute neuromuscular disorder regardless of how long is the rest period. The overall finding, therefore, is that safe work/rest ratios should be 1:1 or lower for work durations up to about 60 min if the development of acute and consequently a chronic disorder is to be prevented. At some point between work duration of 30–60 min even a lengthy rest of 7 h does not effectively block a disorder.

The relevant observation from this study is the preventive effect of a rest period which is of equal duration to the work period up to about 30 min. An equally long rest period seems to allow sufficient time for the viscoelastic tissues to recover to the state where a second work period could be executed without eliciting levels of microdamage that result in an acute inflammation and the associated delayed hyperexcitability of the posterior musculature. A sufficiently long rest period, therefore, could be established as a prominent factor that can prevent or attenuate the development of CTD. Additional support for such an observation is offered by our previous conclusions that six sessions of 10 min work followed by 10 min rest also did not result in any delayed hyperexcitability whereas six sessions of 10 min work spaced with only 5 min rest periods did elicit a delayed hyperexcitability (Courville et al., 2005). Furthermore, allowing 20 min of rest between six 10 min work periods did not result in any additional benefits that could be immediately observed. From an economical standpoint, it is desirable to accomplish as much work as possible within the workday. Unnecessary longer rest periods, therefore will reduce the output of the worker, increase the cost of labor and decrease productivity

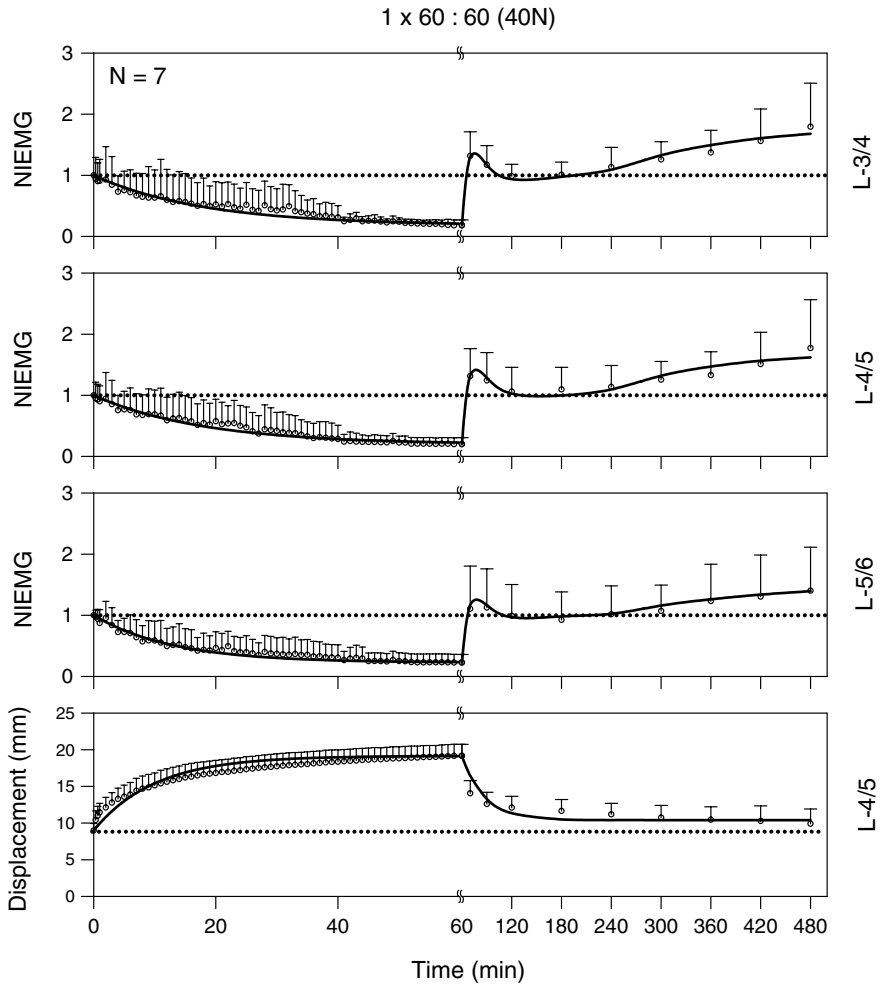


Fig. 4. The mean (± SD) NIEMG of the L-3/4, L-4/5, and L-5/6 multifidus as well as the mean displacement during loading and 7 h recovery for the $1 \times 60{:}60$ group are shown. The solid lines through the data points represent the models developed.

without benefit. Determination of the optimal work/rest ratio however will insure the long-term health of the worker while offering a reasonable efficiency.

The typical response to the three work/rest combinations (30:30 min, 30:10 min, and 60:60 min) consisted of a gradual decrease of EMG with superimposed spasms during the work periods. The 7 h of recovery consisted of an initial hyperexcitability during the first hour followed by a gradual asymptotic increase of EMG to or above its original level. In the experimental group subjected to only 10 min rests, a delayed hyperexcitability was present as compared to the group subjected to 30 min rest. By the fourth hour of recovery, the NIEMG exceeded 1.0 for all of the three lumbar levels in the group subjected to only 10 min rest. At the end of the recovery period, the NIEMG reached 1.542, 1.375, and 1.643 in the multifidi muscles of the three lumbar levels. This corresponds to muscular activity which is 54.2%, 37.5% and 64.3% higher than normal. Indeed, the clinical literature points out that patients with low back disorders display substantially higher EMG levels (Shivonen et al., 1991; Hoyt et al., 1981; Miller, 1985; Haig et al., 1993), supporting the assertion that tissue damage is manifested by higher than normal EMG and was present in the group allowed only 10 min rest and the group subjected to continuous 60 min work. The increased stiffness of the spine due to the increased muscular activity is thought to protect the soft tissue from any additional damage by limiting the range of motion and the associated displacement (or creep).

Interestingly, all experimental groups exhibited the development of creep in parallel with spasms from the multifidus muscles. Spasms are mostly associated with tissue damage. Furthermore, the initial hyperexcitability was also observed in all of the experimental groups. It seems that creep and the associated microdamage in the collagen fibers were present in the response of all the groups, yet the allowance of equally long rest afforded sufficient recovery of these adverse reactions such that longer lasting cumulative damage was avoided as evidenced by the absence of the delayed hyperexcitability and its associated acute inflammation (Solomonow et al., 2003a,b). This observation further strengthens the conclusion that a proper rest period is a dominant factor in preventing CTD.

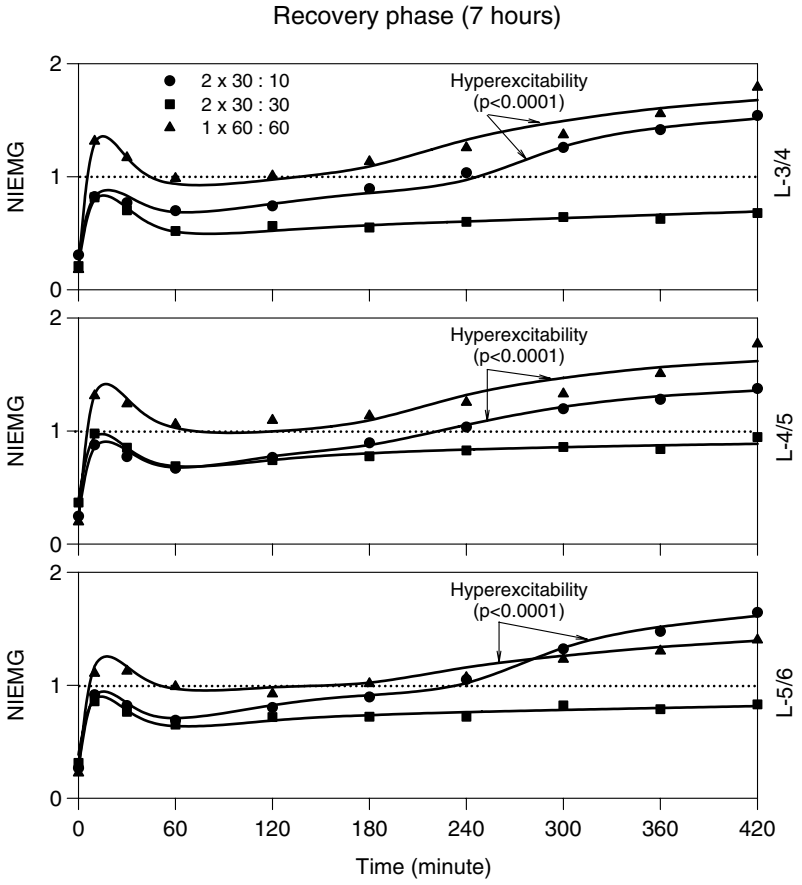


Fig. 5. The mean NIEMG data and the developed models for the 7-h recovery period are superimposed for the three experimental groups. Filled square symbols represent data from the $2 \times 30{:}30$ group; filled circles represent data from the $2 \times 30{:}10$ group and filled triangles represent the $1 \times 60{:}60$ group.

The 1:1 work/rest ratio was optimal as far as preventing the delayed hyperexcitability and inflammation as the initial phase of the CTD while optimizing work output efficiency. This, however, was true only for the range of work/rest durations up to 30 min. When longer duration of 60 min work followed by more than equally long rest was employed, the initial phase of CTD (a neuromuscular disorder as manifested by the delayed hyperexcitability) was present. It seems that as the duration the lumber spine is under load in flexion exceeds a certain threshold period; the microdamage developed in the viscoelastic tissues is apparently excessive to the point that an equally long or longer rest period cannot restore it, and an inflammation sets into the tissues. This leads us to conclude that the microdamage developed in the viscoelastic tissues throughout the static flexion phase is cumulative. As the static flexion is prolonged beyond certain threshold duration, the microdamage level is sufficient to trigger an acute inflammation and therefore not responsive to immediate, equally long rest. Long static flexion periods should be avoided as a matter of principle, as they seem to be damaging the lumbar tissues as a function of time and without being responsive to generous rest periods. It could be concluded that equal work/rest durations are optimal up to some duration between 30 and 60 min, where the length of time under load develops tissue microdamage that could not be offset by equally long rest.

Another issue of importance emerges from the observations above. Microdamage in viscoelastic tissues during static lumbar flexion must be a common occurrence in many daily activities, yet it may have only minor transient and mostly unnoticed effects as long as it is below a given threshold. As the microdamage seemed to exceed a certain level, in a response to overly prolonged activity, an acute inflammation and the associate neuromuscular disorder are triggered in order to heal the affected tissues. Intermediate rest periods, even long ones are not effective in preventing or attenuating the development of inflammation or neuromuscular disorder. From the ergonomic standpoint, identification of the threshold levels of the work duration that triggers excessive tissue microdamage, inflammation and disorder in healthy humans within the various branches of the workforce in industry should be of high importance and priority. In the feline model, the work duration that elicits a neuromuscular disorder not responsive to equally long rest is between 30 and 60 min. Due to various developmental issues, this threshold period may be different in humans, and that needs to be determined.

Recent work with humans, testing the lumbar spine and the knee ligaments demonstrated that static and cyclic

loading/flexion results in spasms during the exposure period and a neuromuscular disorder post-exposure, (Olson et al., 2004; Sbriccoli et al., 2005; Solomonow et al., 2003d; Chu et al., 2003). Therefore one can conclude that while differences may exist between the quadruped feline and biped human, the general principles governing the physiology and biomechanics of CTD development may be identical. Therefore, the central issues that govern the initial development of CTD such as creep, tissue damage, spasms, delayed hyperexcitability and inflammation as well as the risk factors that elicit the process are probably identical. Scaling issues that account for size differences as well as developmental or genetic issues that account for differences in tolerance to work may influence the conclusions developed in this study. That will probably manifest in changes in the exact thresholds of work:rest ratios but not in the fact that overly long flexion periods could not be compensated by rest as a preventive measure for the development of CTD.

5. Conclusions

Based on the results of this investigation, the following contemporary conclusions could be made:

- Static lumbar flexion periods could be performed sequentially without eliciting a prolonged neuromuscular disorder as long as the in-between rest periods are of equal duration to the flexion periods.
- A 1:1 work/rest ratio seems to be an optimal one while considering long term safety and productivity.
- The 1:1 ratio holds for durations up to 30:30 min. At some point between 30 and 60 min even a longer rest than the work duration may not attenuate a disorder.
- The duration lumbar viscoelastic tissue is under tension in elongation is important and when exceeding a certain threshold, an adverse reaction, which is not responsive to rest, results.
- Rest periods, within the ranges and doses described above, maybe a useful risk prevention and attenuation mechanism.

The new conclusions listed above allow new insights into the development and prevention of CTD as well as to structuring optimal work/rest occupational activities. In perspective, some additional information may be required to arrive to a conclusive overview such as the number of repetitions that the 1:1 ratio may hold as risk free and whether large loads may further compromise the 1:1 ratio as risk free.

Acknowledgments

This work was supported by the National Institute of Occupational Safety and Health with Grants R01-OH-04079 and R01-OH-07622. Paola Sbriccoli, MD, PhD was a research fellow from the Department of Human Movement and Sport Sciences, University Institute of Motor Sciences (IUSM) of Rome, Italy.

References

Chu D, LeBlanc R, D'Ambrosia P, D'Ambrosia R, Barrata RV, Solomonow M. Neuromuscular disorder in response to ACL creep. Clin Biomech 2003;18:222–30.

Claude LN, Solomonow M, Zhou B-H, Baratta RV, Zhu MP. Neuromuscular dysfunction elicited by cyclic lumbar flexion. Muscle Nerve 2003;27:348–58.

Courville A, Sbriccoli P, Zhou B, Solomonow M, Lu Y, Burger E. Short rest periods after static lumbar flexion are a risk factor for cumulative low back disorder. J Electromyogr Kinesiol 2005;15:37–52.

Eversull E, Solomonow M, Zhou B-H, Baratta RV, Zhu MP. Neuromuscular neutral zones sensitivity to lumbar displacement rate. Clin Biomech 2001;16:102–13.

Haig A, Weisman G, Haugh L, Pope M, Grouber L. Prospective evidence for change in paraspinal muscle activity after herniated nucleus pulposus. Spine 1993;18:926–30.

Hoogendoorn E, Bongers PM, de et HC, Douwes M, Koes BW, Miedema MC, et al. Flexion and rotation of the trunk and lifting at work are risk factors for low back pain: results of a prospective cohort study. Spine 2000;25:3087–92.

Hoyt W, Hunt H, DePauw M. EMG assessment of chronic low back pain Syndrome. J Am Osteopath Assoc 1981;80:728–30.

LaBry R, Sbriccoli P, Zhou B-H, Solomonow M. Longer static flexion duration elicits a neuromuscular disorder in the lumbar spine. J Appl Physiol 2004;96:2005–15.

Leadbeter W. An introduction to sports induced soft tissue inflammation. In: Leadbeter W, Buckwalter J, Gordon S, editors. Sports induced inflammation. Park-Ridge (IL): AAOS; 1990.

Lu D, Solomonow M, Zhou B, Baratta R, Li L. Frequency dependent changes in neuromuscular response to cyclic lumbar flexion. J Biomech 2004;37:845–55.

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

Miller D. Comparison of EMG activity in the lumbar paraspinal muscles of subjects with and without chronic low back pain. Phys Ther 1985;65:1347–54.

National Academy of Sciences. Musculoskeletal disorders and the workplace. Washington (DC): National Academy Press; 2001.

Olson M, Li L, Solomonow M. Flexion–relaxation response to cyclic lumbar flexion. Clin Biomech 2004;19:769–76.

Panjabi M, Courtney T. High speed stretch of rabbit ACL: change in elastic failure and viscoelastic characteristics. Clin Biomech 2001;16:334–40.

Punnett L, Fine L, Keyserling W, Herrin G, Chaffin D. Back Disorders and non neurtral trunk postures of automobile assembly workers. Scand J Work Environ Health 1991;17:337–46.

Sbriccoli P, Solomonow M, Zhou B-H, Baratta RV, Lu Y, Zhu M-P, et al. Static load magnitude is a risk factor in the development of cumulative low back disorder. Muscle Nerve 2004a;29:300–8.

Sbriccoli P, Yousuf K, Kopershtein I, Solomonow M, Zhou B, Zhu M, Lu Y. Static load repetition is a risk factor in the development of lumbar cumulative musculoskeletal disorder. Spine 2004:2643–53.

Sbriccoli P, Solomonow M, Zhou B, Lu Y, Sellards R. Neuromuscular response to cyclic loading of the ACL. Am J Sports Med 2005;33:543–51.

Shivonen T, Partanen J, Hanninen O, Soimakallio S. Electric Behavior of low back muscles during lumbar pelvic rhythm in low back pain and Healthy controls. Arch Phys Med Rehabil 1991;72:1080–7.

Silverstein B, Fine L, Armstrong T. Hand and wrist cumulative trauma disorders in industry. Brit J Indus Med 1986;43:779–84.

Solomonow M. Ligaments: a source of work-related musculoskeletal disorder. J Electromyogr Kinesiol 2004;14:49–60.

Solomonow M, Zhou B, Baratta RV, Zhu M, Lu Y. Neuromuscular disorders associated with static lumbar flexion: a feline model. J Electromyogr Kinesiol 2002;12:81–90.

Solomonow M, Hatipkarasulu S, Zhou B-H, Baratta RV, Aghazadeh F. Biomechanics and electromyography of a common idiopathic low back disorder. Spine 2003a;28(12):1235–48.

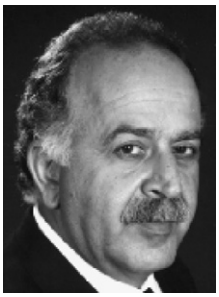
Solomonow M, Baratta RV, Zhou BH, Burger E, Zieske A Gedalia. Muscular dysfunction elicited by creep of lumbar viscoelastic tissues. J Electromyogr Kinesiol 2003b;13:381–92.

Solomonow M, Zhou B-H, Baratta RV, Burger E. Biomechanics and electromyography of a cumulative lumbar disorder: response to static flexion. Clin Biomech 2003c;18:890–8.

Solomonow M, Baratta RV, Banks A, Freudenberger C, Zhou B. Flexion–relaxation response to static lumbar flexion. Clin Biomech 2003d;18:273–9.

Woo S, Aprelena M, Hoher J. Tissue mechanics of ligaments and tendons. In: Kumar S, editor. Biomechanics and ergonomics. Philadelphia (PA): Taylor and Francis; 1999.

Paola Sbriccoli, Ph.D. in Physiopathology of Movement, was born in 1964. In 1986 she graduated "cum laude" at the ISEF (Superior Institute of Physical Education) of Rome. In 1994 she received her Medical Doctor degree from the University of Rome "La Sapienza" and in 1998 she specialized "cum laude" in Sport Medicine. From May 2003 to April 2004 she has attended a research fellowship at the Bioengineering Laboratory (Department of Orthopaedics) of the Louisiana State University Health Sciences Center in New Orleans, Louisiana, USA where she worked on neuromuscular function and dysfunction in spine and knee in humans and animal preparation. Her present appointment is at University Institute of Motor Sciences of Rome, Faculty of Motor Sciences as researcher in Methods and Teaching of Sports Activities. She is ordinary member of the Physiological Society of Italy, the European College of Sport Science, and the International Society for Electrophysiology and Kinesiology. From 2004 she is Member of the Editorial Board of the Journal of Electromyography and Kinesiology. Her main interests in research are non-invasive assessment of muscle damage and repair, linear and non-linear analysis of sEMG signals in healthy humans, and exercise physiology.



Dr. Moshe Solomonow is a Professor and director of the Bioengineering Division and the Musculoskeletal Disorders research Laboratory in the Department of Orthopedics at the University of Colorado Health Sciences Center in Denver. He was a Professor and Director of Bioengineering and of The Occupational Medicine Research Center at Louisiana State University Health Sciences Center in New Orleans, Louisiana from 1983 to 2005. He received the B.Sc., and M.Sc. in Electrical Engineering and the Ph.D. in Engineering Systems and Neuroscience from the University of California, Los Angeles.

He is the Founding Editor of The *Journal of Electromyography and Kinesiology*, and serves on the Editorial Board of several bioengineering and medical journals. He is/was a consultant to the National Science Foundation, National Institute of Health, Center for Disease Control, The Veterans Administration and scientific agencies of several European and Asiatic governments and Canada. He was a council member of the International Society of Electrophysiological Kinesiology, the International Society of Functional Electrical Stimulation, and the IEEE-Biomedical Engineering Society. He published over 120 refereed journal papers on musculoskeletal disorders including: motor control, Electromyography, muscle, tendon, ligament and joint Biomechanics, electrical muscle stimulation, prosthetics and orthotic systems for paraplegic locomotion, and supervised more than 150 engineering, physical therapy, medical students and orthopaedic residents, as well as postgraduate students and fellows from several countries.

He organized the EMG Tutorial Workshop in the ISB Congress, the Canadian Society of Biomechanics, The Human Factors and Ergonomics Society, and The Society for Clinical Movement Analysis, was on the organizing committee of numerous conferences and gave keynote and symposia lectures in many others. He received the Crump Award For Excellence in Bioengineering Research (UCLA), the Distinctive Contribution Award from Delta 7 Society (France), The Doctor Medicine Honoris Causa (Vrije Universitiet, Brussels), The I. Cahen Professorship (LSUHSC) and the 1999 Volvo Award For Low Back Pain Research.

Bing He Zhou (M'89) graduated in 1970 from the Department of Electronic Engineering, University of Science and Technology of China (USTC) in Beijing, China.

From 1970 to 1978, he worked as an Electronics Engineer at the Beipiao Broadcasting Station in Liaoning Province. In 1978, he joined the faculty of the Department of Electronic Engineering at USTC, where he was an Associate Professor of Electronic and Biomedical Engineering and the Vice Director of the Institute of Biomedical Engineering. From 1985 to 1987, he was a Visiting Research Professor in the Bioengineering Laboratory at Louisiana State University Medical Center (LSUMC) in New Orleans, where he worked with the laboratory staff on various studies related to the analysis and control of the neuromuscular system, electromyography, and instrumentation design. Currently, he is a Visiting Research Professor in the Bioengineering Laboratory at LSUMC. His teaching and research interests focus on analog and digital electronics, biomedical electronics, digital signal processing, and microcomputerized medical instrumentation.

He is a Committee Member of the International Union of Radio Science (USRI), the Commission of Electromagnetics in Biology and Medicine (Commission K), and the Chinese Biomedical Electronic Society. He is also a Senior Member of the Chinese Electronic Society, as well as a member of the Chinese Biomedical Engineering Society, the Chinese Computer Society, and the IEEE/Engineering in Biology and Medicine Society. He received the Zhang Zhongzhi Award for excellent teaching and research activities at USTC in 1989, and first-place awards for most outstanding academic paper from the Chinese Biomedical Electronic Society (1991) and the Anhui Biomedical Engineering Society (1992).

Dr. Yun Lu received his medical degree from Xian Medical College in China in December of 1982. He was an orthopaedic resident from 1983 to 1986; chief resident from 1986 to 1987; and attending orthopedic surgeon from February 1988 to December 1988, at West Capital Hospital of the Fourth Military Medical University, Xian, China. He was a post-doctoral fellow from 1988 to 1989 at the Department of Orthopaedic Surgery, School of Medicine, Johns Hopkins University, Baltimore, MD, and a fellow in the Department of Orthopaedic Surgery at Louisiana State University in New Orleans, since 1989. He is a Research Professor in the Bioengineering Laboratory at LSUHSC.