Muscular dysfunction elicited by creep of lumbar viscoelastic tissue

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Abstract

The biomechanics, histology and electromyography of the lumbar viscoelastic tissues and multifidus muscles of the in vivo feline were investigated during 20 min of static as well as cyclic flexion under load control and during 7 h of rest following the flexion. It was shown that the creep developed in the viscoelastic tissues during the 20 min of static or cyclic flexion did not fully recover over the 7 h of following rest. It was further seen that a neuromuscular disorder with five distinct components developed during and after the static and cyclic flexion. The neuromuscular disorder consisted of a decreasing magnitude of reflexive EMG from the multifidus upon flexion as well as of superimposed spasms. The recovery period was characterized by an initial muscle hyperexcitability, a slowly increasing reflexive EMG and a delayed hyperexcitability. Histological data from the supraspinous ligament demonstrate significant increase ($\times 10$) in neutrophil density in the ligament 2 h into the recovery and even larger increase ($\times 100$) 6 h into the recovery from the 20 min flexion, indicating an acute soft tissue inflammation.

It was concluded that sustained static or cyclic loading of lumbar viscoelastic tissues may cause micro-damage in the collagen structure, which in turn reflexively elicit spasms in the multifidus as well as hyperexcitability early in the recovery when the majority of the creep recovers. The micro-damage, however, results in the time dependent development of inflammation. In all cases, the spasms, initial and delayed hyperexcitabilities represent increased muscular forces applied across the intervertebral joints in an attempt to limit the range of motion and unload the viscoelastic tissues in order to prevent further damage and to promote healing.

It is suggested that a significant insight is gained as to the development and implications of a common idiopathic low back disorder as well as to the development of cumulative trauma disorders.

1. Introduction

The motor control of lumbar motion, like that of most other single joints, consists of a closed loop feedback system[33,44] depicted schematically in Fig. 1. The forward segment of this control system consists of motor neurons and their associated muscles energizing skeletal segments to develop a prescribed motion or posture in terms of angle, position, torque, velocity, etc. The control inputs to the forward segment are derived from pyramidal signals descending from the motor cortex as well as complex reflexes derived from the cerebellum and other supraspinal sources. The feedback segment of the control system consists of the traditional inputs from the proprioceptive (muscle senses), kinesthetic and tactile senses as well as various spinal reflexes. One of the least investigated of these feedback loops is that from afferents in viscoelastic tissues (e.g., ligaments, discs and capsules). Four types of afferents: Golgi, Ruffini, Pacinian and bare nerve endings populate the ligaments of the extremity joints [36,37,47,60] and of the spine [15,21,35,39,59]. In the spine, afferents are also present in the discs and in the capsules [15,21,35] Stretch, load or electrical stimulation applied to the ligaments, discs or capsules was shown to elicit a reflex activation of muscles [18,19,27,45,47,51,55] that have excitatory stabilizing effect on the joint in certain conditions

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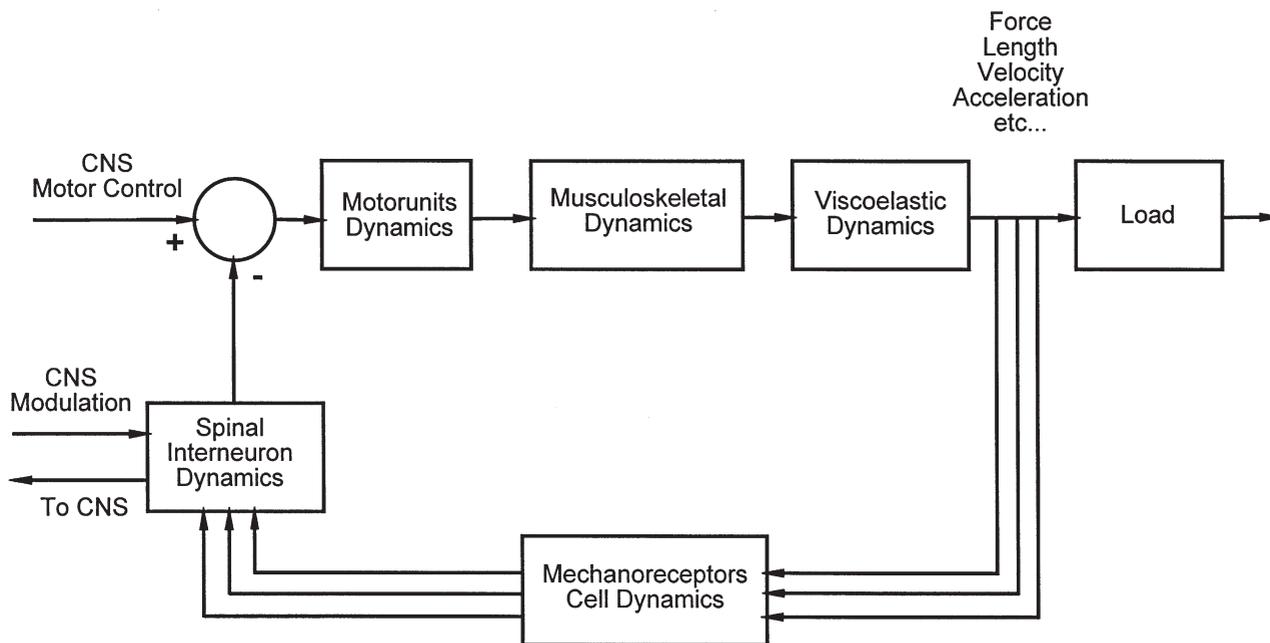*E-mail address:* msolom@lsuhsc.edu (M. Solomonow).

Fig. 1. A simplified feedback control system of an intervertebral joint of the spine.

[27,45,47] and inhibition of muscle activity in other conditions [24]. From the nature of the afferents found in the viscoelastic tissues, it is also expected that in addition to functioning as a stabilizing reflex feedback sensors, they may also contribute to perception.

Viscoelastic tissues are known to behave in a complex manner with some well recognized characteristics. When subjected to load, ligaments and discs develop creep over time [1,8,12,14,23,31,56] and the creep requires much longer to recover than the duration of the stimulus that caused its development [5,8,31]. Similarly, elongation of ligaments is associated with tension–relaxation which also requires periods of recovery which are much longer than the duration of the elongation that caused its development [10,20,49]. Furthermore, cyclic loads applied to viscoelastic tissues are associated with progressively increasing hysteresis over time [46]. Recent experimental, clinical and epidemiological reports point out that viscoelastic tissues subjected to static or cyclic loads over time are subjected to micro-damage that may alter the functional properties of the tissues and lead to disorders [2,26,41,43,53]. Static or cyclic loads applied over time to ligaments, discs, tendon or capsular tissues, therefore, may be a source of viscoelastic tissue dysfunction. Since reflexive activation of muscles is triggered by afferents within such tissues, it is conceivable that a neuromuscular disorder may also be associated with dysfunction of the viscoelastic tissues.

It is the objective of this report to describe some neuromuscular disorders associated with application of static and cyclic loads to the lumbar viscoelastic tissues. In an attempt to follow classical control theory techniques used to identify the functional properties of a given component in a closed loop system, static and cyclic inputs were applied to the feedback components (viscoelastic tissues) while assessing system output [7]. The hypothesis set, is that static or cyclic loads applied to the lumbar viscoelastic tissues over a given period of time will elicit a neuromuscular disorder in some paraspinal muscles.

The following sections describe a series of experiments aimed to test the hypothesis set above. The experiments were performed using a feline model. This allows invasive procedures as well as the isolation of specific mechanical inputs to the feedback segment from other inputs (supraspinal control, vision, audition, etc. . . .) active in behaving humans, which may cloud the results. The feline model, therefore, is enriching the clear insight into the dynamics of the viscoelastic tissues feedback components and their role in the development of low back disorders. The detailed experimental set-up and analytical technique are given in our previous reports [4,48,49,50].

2. Neuromuscular disorders associated with static loading

Feline preparations, anaesthetized with chloralose were subjected to static lumbar flexion via a stainless steel hook that was inserted around the L-4/5 supraspinous ligament and loaded with a material testing system MTS Bionix 858 in load control mode. The applied loads were tested in preliminary experiments to yield strains of the ligaments within their physiological range [34,58], while developing moderate lumbar flexion. Wire EMG

electrode pairs were inserted into the multifidus of L1/2–L-6/7 levels. Several load levels were applied in order to assess if the load level is a dominant factor in the development of dysfunctions.

A typical response of the EMG from the multifidus muscles as well as the associated displacement of the spine to several load magnitudes for a 20-min long static lumbar flexion is shown in Fig. 2.

The four columns shown in Fig. 2 are typical responses from four different preparations, each loaded at a different magnitude ranging from 20 N [at left] to 70 N (at right). One typical response of the multifidus muscles is a pronounced EMG discharge as soon as the viscoelastic tissues were loaded into flexion. As time went on, the EMG decreased in an exponential fashion and settled at some steady state level near 45–50% of its initial amplitude. Fig. 3 provides the mean ± SD of the Normalized Integrated EMG (NIEMG) throughout the 20-min static loading period. The EMG response of the first second was integrated, and was used as a basis

for normalizing following windows. The data shown in Fig. 3 include large standard deviations as well as several obvious deviations from an exponential like decrease, the source of which is described in the following paragraphs. The decrease in EMG represents decrease in muscle force. Since the lumbar spine was loaded into anterior flexion, the multifidi were part of the muscles controlling the accuracy and stability of the movement. Reduction in the EMG from the multifidi, therefore, represent decrease in overall controllability and stability of the movement and increased risk of exposure to injury [11,25,29,30,45]. In essence, in the first 2–3 min the muscles provided normal function and force to stabilize the movement and the joints, but this beneficial and necessary function decreased thereafter, and remained low as it reached the steady state level.

A second typical response of the multifidi muscles during the 20-min static load application were unpredictable spasms superimposed on the reflexive EMG. The spasms were present in every preparation regardless of
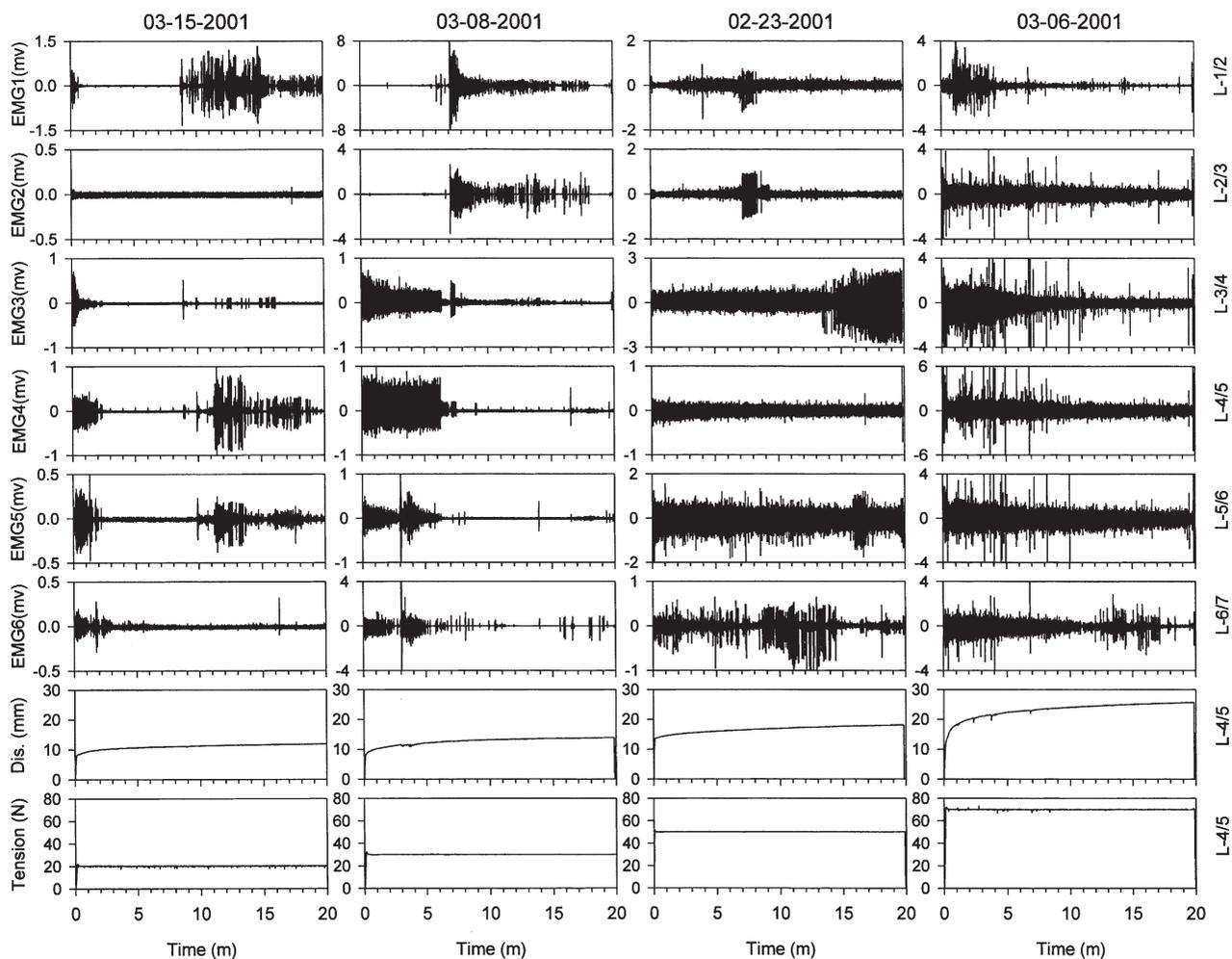

Fig. 2. Typical EMG response to several static load levels applied over 20-minutes. Note the spasms in L-1/2, L-3/4, L-4/5 and L-5/ after the 9th minute in Column 1. In Column 2, the spasms in L-3/4 to L-6/7 diminish just as spasms in L-1/2 and L-2/3 start. The load applied in each column is shown in the bottom panel.
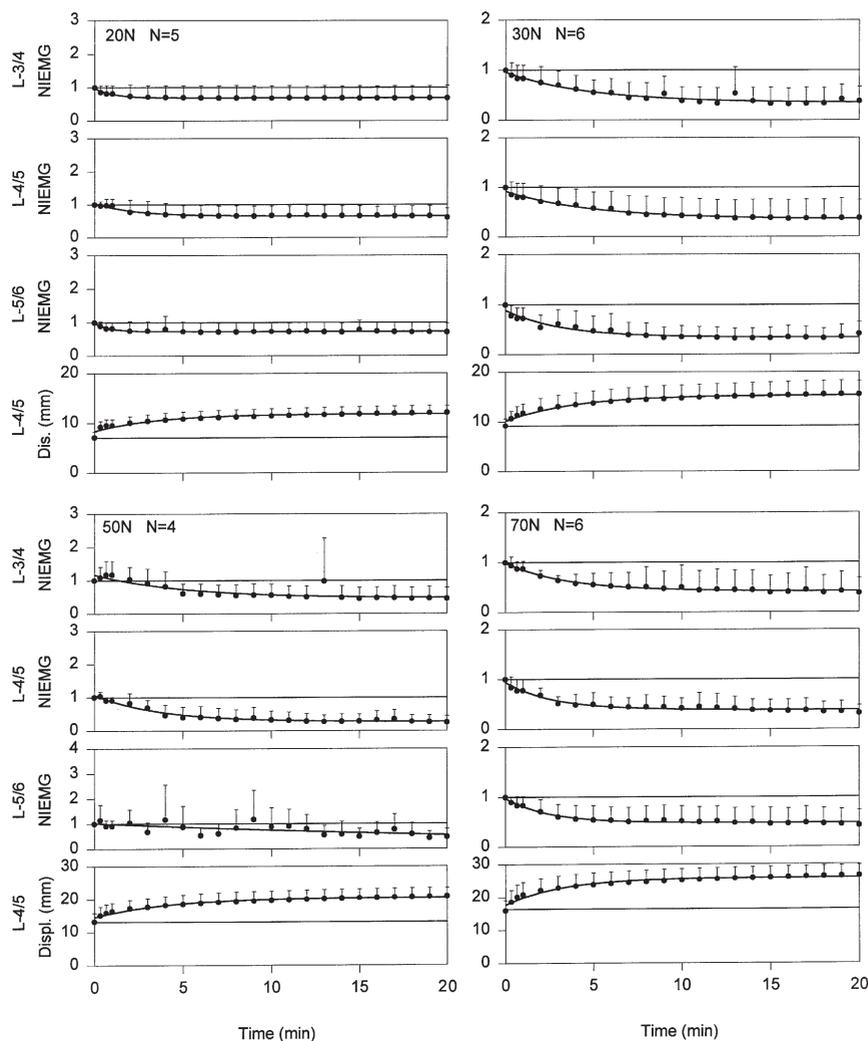
Fig. 3.   Mean Normalized Integrated EMG and displacement throughout the 20 min of static loading of several preparations exposed to the same load magnitude. The solid lines through the means represent the model developed.

the load magnitude applied. In some preparations the spasms appeared in all levels of the multifidus muscles at the same time whereas in other preparations the spasms were not synchronized in all levels. In other preparations, yet, appearance of spasms in one or two levels was accompanied with inhibition of spasms in other levels. Furthermore, the timing of the spasms was unpredictable as well, appearing sometimes early on, sometimes in mid-session and sometimes late in the loading session. In preparations subjected to the highest load magnitudes, the spasms were often characterized by large amplitude spikes throughout the loading session.

The random and unpredictable appearance of the spasms had the effect of increasing the standard deviation of the means of the NIEMG patterns shown in Fig. 3, and sometimes resulted in significant modification of the mean value such that deviation from an exponential like decrease were observed. Since it is impossible to filter out the spasms, they were left in the original recordings when the mean data was calculated, yet the gen-

eral pattern of exponential like decrease in the reflexive EMG was still obvious from Fig. 3.

Spasms and elevated EMG activity are long known as muscular response to tissue damage and the associated pain [9,13,17,32,35,40,42]. More specific evidence points out that spasms are the muscular response to tissue damage in an attempt to maintain some stability to the joint and prevent further damage [28]. Lund and co-authors pointed out that the antagonist muscle is often activated by spasms to an increased level in order to reduce the velocity and range of motion of attempted movement, limiting additional damage. Inhibitory influences of the agonist muscle were also associated with pain and tissue damage, further limiting the movement and additional damage. Indeed, recent work comparing healthy controls to low back pain patients found marked increase in antagonist to agonist discharge ratio in the patient group [57]. What is important to note is that the damage or the pain did not originate from the muscles, but that the muscles were recruited to limit the additional damage (and probably pain) in the viscoelastic tissues.

As pointed out earlier, the mechanical/histological literature points out that cyclic or static load applied to ligaments results in micro-damage in the structure of the tissue as well as changes in its mechanical properties [2,26,41,54]. It is therefore suggested that the EMG spasms are the result of micro-damage in the tissues of the ligaments, discs and capsules of the lumbar spine. The fact that creep developed in these tissues over time points out that changes in their mechanical properties were indeed present. Furthermore, the recovery of the creep with rest, as will be discussed in a following section, demonstrated that the changes in the mechanical properties of the viscoelastic tissue lasted more than 7 h and that the tissues were indeed inflamed. Since bare nerve endings, which are pain mediators, exist in the viscoelastic tissue, it is conceivable that they triggered the spasms and probably pain/discomfort sensation.

The recovery period following the 20 min of static lumbar flexion allowed the viscoelastic tissues of the lumbar spine to rest and attempt to restore their original mechanical properties. Short test periods of 10 s static loads were applied throughout the rest period in order to assess the recovery of the tissues mechanical properties as well as the corresponding EMG from the multifidus muscles. Fig. 4 presents a typical recording of EMG and flexion displacement in response to short load tests throughout the recovery period, whereas Fig. 5 provides the mean of EMG and flexion displacement of all the preparations tested at the same load.

Three distinct features characterize the EMG behavior during the recovery period; initial hyperexcitability, gradual increase in EMG towards its original resting level and delayed hyperexcitability.

During the first hour of rest, immediately following 20 min of static flexion, the EMG demonstrated a sharp increase in amplitude upon brief flexion tests followed by a fast decrease in amplitude to a level close to near that recorded at the end of the 20 min of flexion. This hyperexcitability of the multifidus to flexion was over within the first hour.

It should be also noted that the majority of the recovery of the creep developed in the flexion displacement also occurred during the first hour. The hyperexcitability in the EMG and the simultaneous large recovery of the creep in the viscoelastic tissues suggest that the muscles increased their level of activity in order to protect the viscoelastic tissues from further damage by limiting the movement or compensating for the lost tensions in the ligaments etc. Once a substantial (but not complete) recovery of the creep took place, the initial hyperexcitability diminished.

The second feature evident in the EMG of the recovery period is a gradual, exponential like increase in the EMG as the creep in the displacement is slowly continuing its recovery. This exponential recovery in the EMG was observed also when the lumbar spine was subjected
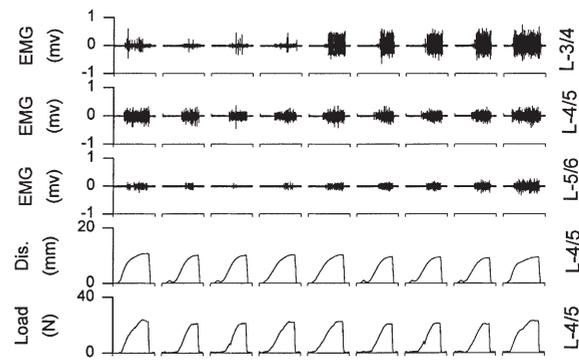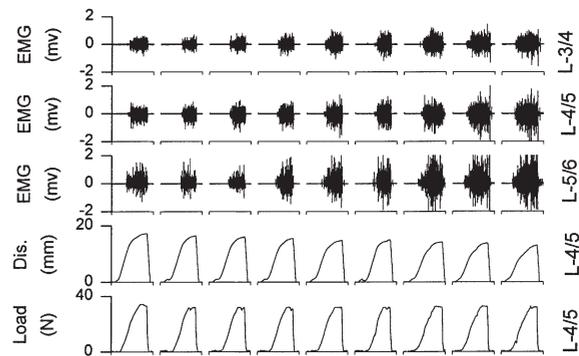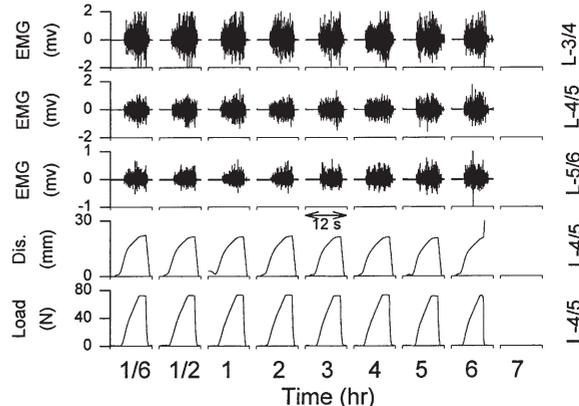

Fig. 4. Typical recordings of EMG from the multifidi as well as flexion displacement and test loads during 7 h of rest after the 20 min of static loading.

to static elongation (as opposed to the static load used in this experiment).[21] In essence, as the creep in the stretched viscoelastic tissues continued to recover, the non-nociceptive mechanoreceptors in the tissues became more sensitive to the applied load, and responded with gradually increased EMG over time. As the EMG from the muscles increased, the forces available from their contraction increased, lending more stiffness and stability to the intervertebral joints. It is important to note that the creep developed in only 20 min of static flexion required much longer to recover and the same applies to the EMG. This issue is important, as it can shed more
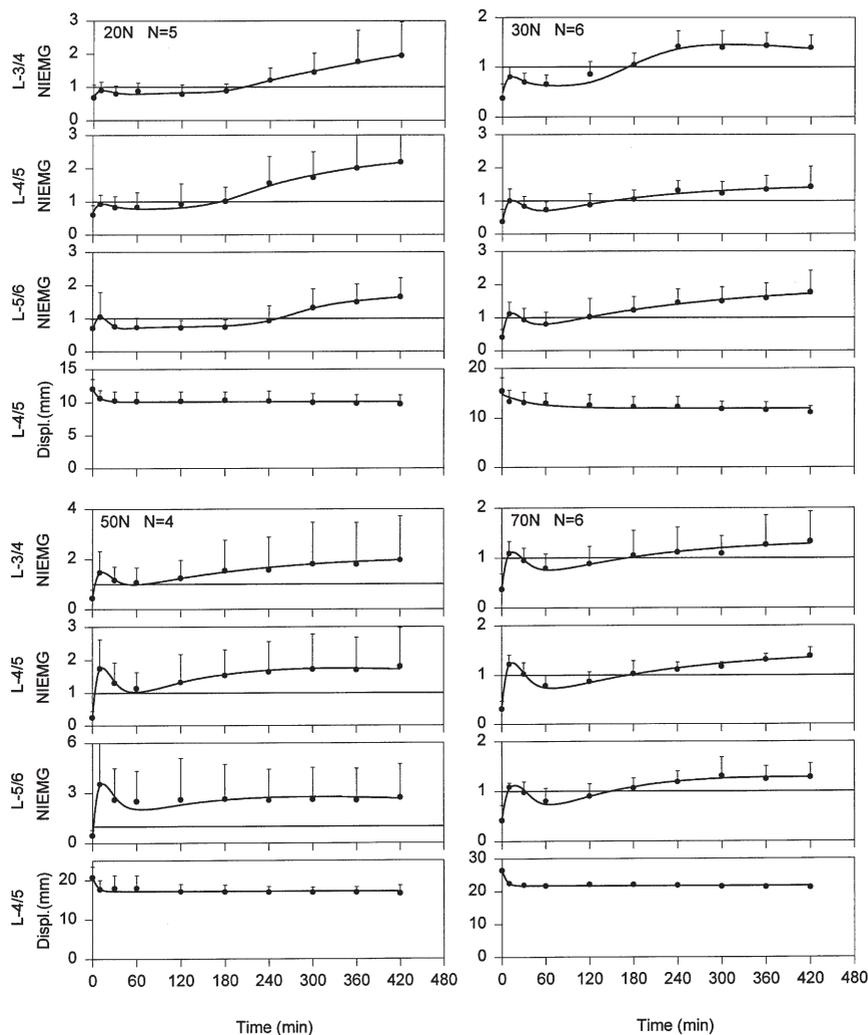
Fig. 5. Mean EMG from the multifidi as well as mean flexion displacement from three groups of preparations during the 7 h rest period after static flexion. The solid lines through the means represent the developed model.

light on the development of cumulative trauma disorders in workers exposed to long daily sessions of static flexion [such as bricklayers, mechanics, roofers, etc.). In such conditions there is not enough time available for the creep developed in one day's work to fully recover before the next day's work begins with residual creep. At the end of the second day of work, there is more creep in the tissues, and that process continues with each additional work day. At the end of a work week, significant amount of creep is accumulated with the associated laxity in the joint and the decreased stability.

The third feature obvious in the recovery data of Fig. 5 is that the creep in flexion displacement did not fully recovery after 7 h of rest and that the EMG after 5–6 h was substantially higher than the initial EMG recorded at the beginning of the 20 min of static flexion. In some preparations the EMG at the 6 or 7 h was 2–3 times larger than the initial EMG at the beginning of the 20 min of static flexion. It is evident that a second, delayed hyperexcitability of the muscles is associated with static

lumbar flexion some 6–7 h after the flexion was completed.

The explanation of this phenomenon emerges from the established facts that prolonged loads or stretch of viscoelastic tissues result in micro-damage. The orthopaedic literature recognizes such stretch damage, as a sprain, is always associated with some degree of inflammation [41]. The development of inflammation in damaged viscoelastic tissue requires time, since inflammatory cells such as neutrophils are mobilized to the affected area via the circulatory system [22]. As time elapses, neutrophils migrated into the collagenous tissues from the small blood vessels which supply them, and after several hours a full inflammatory condition develops, including elevated temperature and edema. At this point it is not clear if the hyperexcitability of the musculature is responding to the inflammatory conditions or to the micro-damage in the tissue. Yet, the fact that the initial hyper excitability was transient, giving rise to a secondary, delayed hyperexcitability, lends support to the explanation that it is an inflammation triggered feature.

In several preparations the peak of the delayed hyper-excitability was 5–6 h into the recovery, followed by a decreasing EMG activity thereafter, indicating that this is a transient phenomenon. This issue will be further discussed in a following section.

3. Neuromuscular disorders associated with cyclic loading

Certain occupational activities in which a movement is repeated many times over time are designated as repetitive motion. An example of such workers include warehouse workers engaged in loading / unloading a truck full of boxes; assembly line workers engaged in flexing into a car and installing a part and other similar industrial activity. Epidemiological surveys point out that the occurrence of neuromuscular disorders in such worker groups is up to ten times that reported from the general population [16,38,43]. Silverstein et al. [43] also pointed out that the occurrence of complaints was related to the frequency in which such movements were performed as well as the duration over which repetitive movements were executed. Evidently, this type of activity is costly in terms of lost work, disability and medical expenses.

It was chosen to employ cyclic loading of the lumbar spine, also in a load control mode, in order to simulate repetitive flexion. This loading mode also allows the control of the frequency in order to assess its impact on the disorder elicited. Initially, it was thought that cyclic flexion applied to the lumbar spine may not result in a disorder as complex or as severe as that seen in static flexion since the full load is applied each time followed by a half cycle of reduced load. This, however, was proven to be incorrect.

Feline preparations were used in a protocol very similar to that applied in the static flexion experiments described above. The load applied, however, was replaced by a sinusoidal wave at 0.25 Hz. This frequency was chosen as it simulated a flexion and then extension to resting position within 4 s, a relatively moderate frequency. Other frequencies are currently being tested in our laboratory in order to assess the impact of this factor on the elicited disorder.

Fig. 6 shows a typical reflexive multifidus EMG, lumbar displacement and the associated cyclic loading applied over a 20-min period followed by 7 h of rest. It was chosen to use 20 min of cyclic flexion and 7 h of rest as it will allow comparison with identical periods of static flexion as described earlier.

Fig. 7, showing the mean data from all the preparations demonstrates that the EMG and displacement patterns elicited are similar to that of static flexion.

The five disorder components observed in the static flexion experiments were also present in the data col-lected from the preparations subjected to cyclic flexion. During the 20 min of cyclic flexion the reflexive EMG from the multifidus decreased exponentially to 55–60% of its initial value. Unpredictable spasms were also recorded in the multifidus. The spasms were present in nearly all the preparations tested, indicating that cyclic loading also inflicts micro-damage in the collagenous tissues of the lumbar spine. In Fig. 8, the effect of sustained spasms on stiffening the spine is shown. A burst of spasms on the 11th minute seem to stiffen the spine and cause a sharp reduction in displacement and at the same instant to increase the load. Since the test apparatus (MTS) was load controlled, it compensated within one cycle, and brought down the peak load to the prescribed 40 N. The recovery period was characterized by initial and delayed hyperexcitabilities superimposed on the exponential increase in NIEMG towards its original level. It is important to note that the mean NIEMG data from the set of preparations subjected to the lowest load of 20 N shows that the delayed hyperexcitability peaked in the 4–5 h of rest and started decreasing thereafter. The data from the populations subjected to 40 or 60 N peak loads shows that the delayed hyperexcitability is still in progress, without evidence of peaking followed by a decrease.

This issue is important, as it provides an observable evidence that the load magnitude is related to the intensity or severity of the disorder developed by the cyclic flexion. Evidently, lower loads result in less micro-damage in the viscoelastic tissues as well as shorter duration over which the delayed hyperexcitability affects the musculature.

Analysis of variance with repeated measures was applied to the data in order to assess if the load level indeed impacted the EMG and displacement, and therefore the severity of the neuromuscular disorder. The results indicated that statistically significant differences ($P < 0.0001$) existed amongst the loads. Similarly, the displacement was significantly different for different loads. Larger loads resulted in larger initial displacement as well as larger creep. Additional evidence of the impact of the load level on the recovery features were also observed from the time constants dominating the different components of the model which is discussed in the next section.

Overall, one can confirm the epidemiological data which point out that larger loads increase the risk factor associated with repetitive work. Ongoing work in the Occupational Medicine Research Center is exploring the effect of the frequency at which cycling loading is performed on the elicited neuromuscular disorder.

4. The neuromuscular disorder model

In order to model the multi-factorial neuromuscular disorder described in the previous sections it is necessary
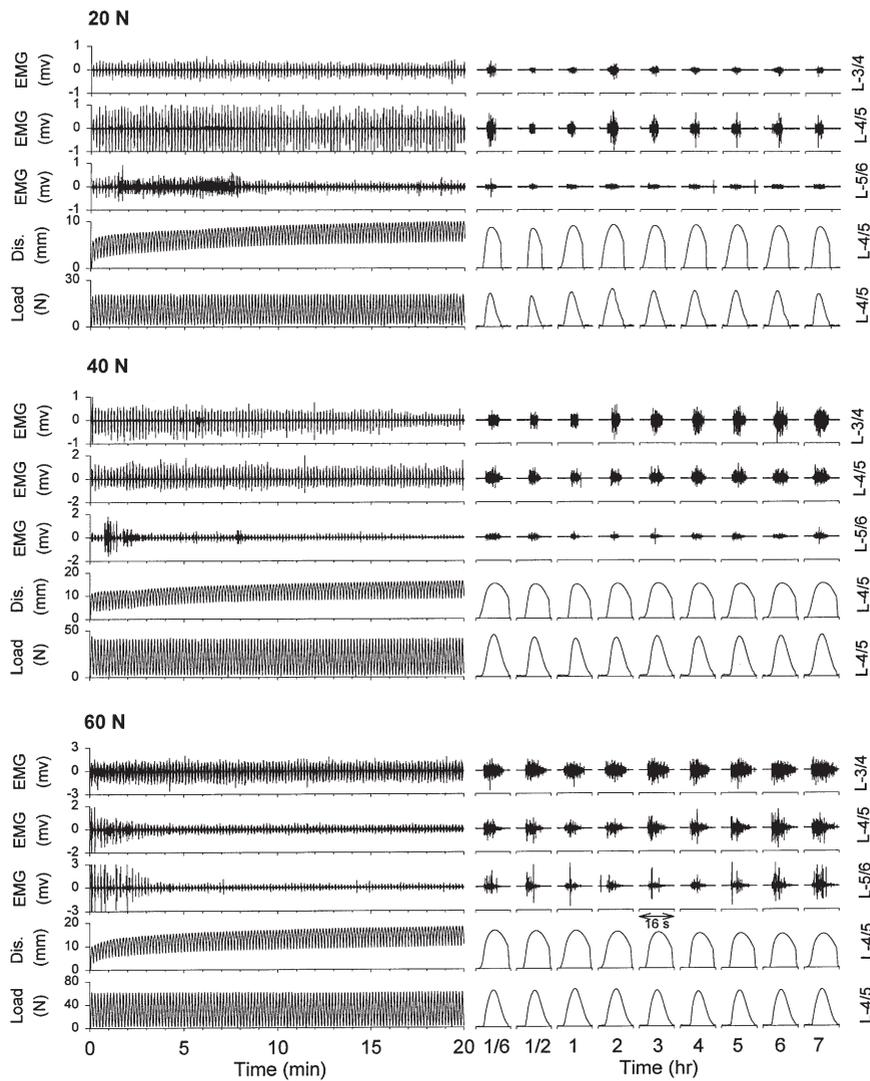
Fig. 6.   Typical recordings of EMG and displacement when cyclic flexion was applied to the lumbar spine followed by 7 h rest.

to consider its various components; the spasms and gradual decrease of EMG during the static flexion as well as the initial and delayed hyperexcitabilities and the gradual recovery of the EMG during the rest period. Spasms, being random and unpredictable phenomena can not be quantified analytically such that one can anticipate their timing, duration, amplitude etc. It is, therefore, left out to a qualitative description. The remaining four features, however, lend themselves to a quantitative description.

The gradual decrease of EMG with flexion was fitted with an exponential term primarily due to the viscoelastic tissues within which the afferents are found. The classical creep response of viscoelastic tissues to load is exponential and one can, therefore, anticipate that the response of the afferents and muscles to load over time will be exponential as well.

The model structure for NIEMG and actuator displacement in the loading period is similar to the one developed by Solomonow et al. [4,48,49] which takes

the form shown in Eqs. (1) and (2), respectively. For the NIEMG:

$$\text{NIEMG}(t) = A\text{e}^{-t/T_1} + \text{NIEMG}_{ss} \tag{1}$$

where,

A = exponential component initial amplitude (unitless)
T_1 = exponential decay time constant in minutes
NIEMG_{ss} = steady state NIEMG amplitude (unitless)
t = time.

The displacement also followed an exponential model:

$$\text{DISP}(t) = D_0 + D_L(1-\text{e}^{-t/T_2}) \tag{2}$$

where,

$\text{DISP}(t)$ = lumbar flexion displacement as a function of time (in millimeters)
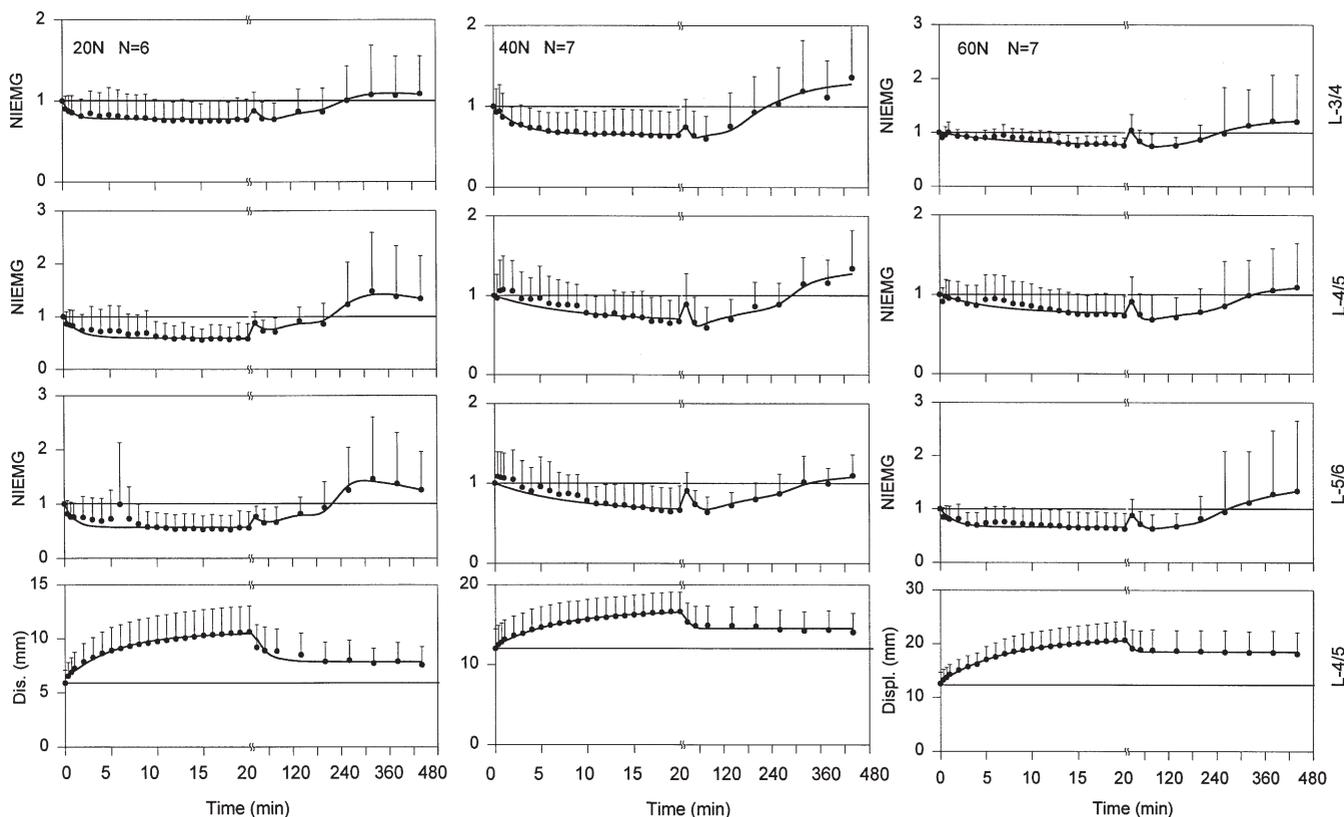
Fig. 7. The mean NIEMG and displacement from three groups of preparations each subjected to different cyclic load. The solid lines through the means represent the model derived.
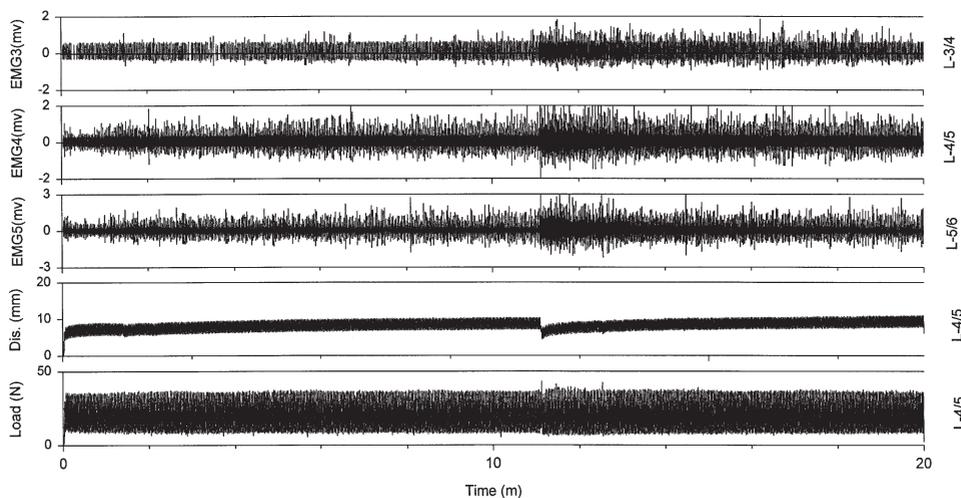

Fig. 8. A typical response to cyclic loading of 40 N peak demonstrates the effect of a burst of spasms in the 11th minute on the displacement and load. The spasms increase the stiffness of the spine which cause a sharp decline in displacement. The increased resistance of the spine to flexion also gave rise to simultaneous increase in the peak load. Since the loading apparatus was load controlled, it compensated for the change within one cycle, and brought the peak load to its original level of 40 N.

D_0 = elastic component of displacement (in millimeters)
D_L = visco - elastic component amplitude of displacement (in millimeters)
T_2 = time constant (in minutes)

t = time.

Similarly, exponential models were chosen to describe the NIEMG and displacement during the 7 h recovery period. The model for the displacement was:

$$\text{DISP}(t) = D_c + D_R e^{-t/T_3} \qquad (3)$$

where

D_c = displacement at the end of the 20-min loading (in millimeters)
D_R = recovery of the creep over 7 h rest (in millimeters)
T_3 = recovery time constant (in minutes).

For the NIEMG, the model format was:

$$\text{NIEMG}(t) = E(1 - e^{-t/T_4}) + tBe^{-t/T_5} + \qquad (4)$$

$$C(t - Td)e^{-(t-T_d)/T_6} + \text{NIEMG}_{ss}$$

where

$E(1 - e^{-t/T_4})$ represents the steady state recovery component
tBe^{-t/T_5} represents the initial transient hyperexcitability component
$C(t - Td)e^{-(t-T_d)/T_6}$ represents a delayed transient hyperexcitability ('morning after'), for $t > T_d$
NIEMG_{ss} represents the residual response at the end of 20-min constant load (unitless)
B, C and E are unitless.

In this model, the constraint of $E + \text{NIEMG}_0 = 1$ is used to insure that full recovery results in a normal (unity) response.

Parameters for the specific models for static and cyclic flexion were obtained by fitting the specific model to the corresponding experimental data using the Marquardt–Levenberg non-linear regression algorithms. Models fitted to the cyclic and static EMG and displacement data are superimposed on the means of the corresponding experimental data in Figures 3, 5 and 7.

The time constants developed for the models above further support that increased loads require longer recovery periods. For cyclic loading, for example, the time constant T_4 dictating the exponential recovery of the reflexive EMG towards its initial level increases from 130 to 240 and to 330 min for loads of 20, 40 and 60 N, respectively. Similarly, the time constant T_6 which describes the exponential decay of the delayed hyperexcitability increases from 113 to 240 and to 290 min for loads of 20, 40 and 60 N, respectively. This implies that the hyperexcitability and probably its associated inflammation will last much longer if one lifts larger loads for the same duration and at the same number of cycles.

The time constants T_d which describe the initiation of the hyperexcitability or its associated inflammation does not change appreciably as a function of load magnitude. A narrow range of 203–216 min was determined by the model for loads of 20–60 N. This is not surprising if one recalls that an inflammatory reaction to tissue damage is dependent on circulatory mechanics which transports neutrophils to the affected tissue via its blood vessels. That reaction time is most likely fixed or limited by circulatory and metabolic factors and not by the extent of the damage in the tissue.

Similarly, the time constant T_5 which governs the exponential associated with the initial hyperexcitability did not exhibit any appreciable changes in response to increased load magnitude, ranging from 7.3 to 10 min. The majority of the creep recovery was in the first hour for all load magnitudes. Since the initial hyperexcitability was responding to the lost forces in the stretched out viscoelastic tissues, which lasted for an hour, then it is conceivable that this transient muscular response will be present only during that hour.

5. Inflammation in the ligaments

The delayed hyperexcitability was explained with the development of inflammation in the lumbar viscoelastic tissues. To date, inflammatory responses of ligaments and tendons were demonstrated after prolonged periods of activity, mostly weeks. Carpenter [2] and Soslowsky [54] developed an in vivo model of rats subjected to down hill running on a treadmill for one hour a day, five days a week for several weeks. Tendon tissue in the shoulder exhibited inflammatory responses which were more intense as the length of exposure to exercise increased. They designated the inflammation as an 'overuse' response. To date, demonstration of inflammation elicited after a short period (20 min) of exposure to static or cyclic loading of viscoelastic tissues is not available. Furthermore, the electromyographic responses to viscoelastic tissue inflammation was also an issue that was not addressed before, and that motivated an exploration into this subject.

Using the same experimental procedures described above, feline preparations were subjected to 20 min of static flexion followed up by 6 h of rest. In one group, the supraspinous ligament of the L-4/5 motion segment was harvested at the end of the 20 min of static loading. In a second group, the L-4/5 supraspinous ligament was harvested after 20 min of static flexion followed by 2 h of rest. In a third group, the ligaments were harvested after 20 min of static flexion followed by 6 h of rest. A control group was also used, from which the L-4/5 supraspinous ligament was harvested without being exposed to flexion or any other loading. The ligaments were fixed in 10% zinc formalin and slides (stained with hematoxin and eosin) were prepared for histological examination for the presence of neutrophils. The presence of elevated number of neutrophils/mm^2 is the standard pathologic test for inflammation. Neutrophils/mm^2 were calculated in each specimen. The mean neutrophil count in the control group was 37/mm^2. In the group

tested immediately after the 20 min of static flexion, the mean neutrophil count was 36/mm^2. The preparations allowed to rest for 2 h past the static flexion exhibited a mean neutrophil count of 160/mm^2, a four times increase over controls. Finally, the group which was allowed to rest for 6 h following the 20 min of static flexion exhibited mean neutrophil count of 4172 neutrophils/mm^2, over 100 times the count found in controls and in the group tested immediately after the static flexion. Fig. 9 provides typical slides of a supraspinous ligament after 6 h rest, showing large infiltration of neutrophils from the walls of a small blood vessel, as well as large density of neutrophils already deposited within the tissue. A specimen from a control ligament is also shown, having only few spontaneously appearing neutrophils.

The histological data suggest that inflammation was not present during the 20 min of static flexion. It further points out that the inflammatory process probably begins immediately after the mechanical stimulus to the viscoelastic tissues was removed. Two hours after the static flexion the neutrophil count was 160/mm^2, four times that of controls.

The presence of a full-scale inflammation in the supraspinous ligament, and its gradual intensification over the rest period can now confirm that the delayed hyperexcitability is associated with, if not directly elicited from the inflammation. It should be noted again, that the damage and inflammation were in the viscoelastic tissues, not in the muscles, yet the muscles responded to the deficit with hyperexcitability. The increased activity of the muscle could be aimed at stiffening the spine and limiting or preventing further micro-damage or aggravation of the viscoelastic structures.

Finally, the peaking of the EMG at 5–6 h of rest and the following decline in several preparations suggest that the phenomena is transitory, where the inflammation and hyperexcitability would gradually decrease with rest. The models developed predict that reduction in EMG to normal levels can occur within 48 h of rest.

6. Discussion

There are numerous types of idiophatic low back disorders. The common denominator of this class of disorders is that routine diagnostic procedures fail to identify their source or their full implications which results in sub-optimal treatment. One of the most common of these disorders is familiar to most readers regardless of their profession or daily activities. A simple session of working in a garden to plant a new bed of flowers which may require a period of static lumbar flexion normally elicits a sensation of gradually intensifying pain or strong discomfort as time goes by. At some point the work is stopped. As one bends over to pick up tools left on the floor, the flexion is associated with great discomfort and stiffness in the lumbar musculature. A rest following a shower provides significant relief. Yet, getting out of bed the next morning is accompanied with pain or discomfort and severe stiffness of lumbar musculature with difficulties in bending over to put on shoes or picking up low objects. This condition may last for 1–3 days after which it is forgotten as all discomfort/ pain, stiffness and limited motion disappear.

It has been commonly thought that muscle fatigue or overuse is the source and cause of the problem. This explanation is hard to accept as deep flexion is associated with the flexion–relaxation phenomenon, where the muscles are deactivated and therefore could not fatigue.
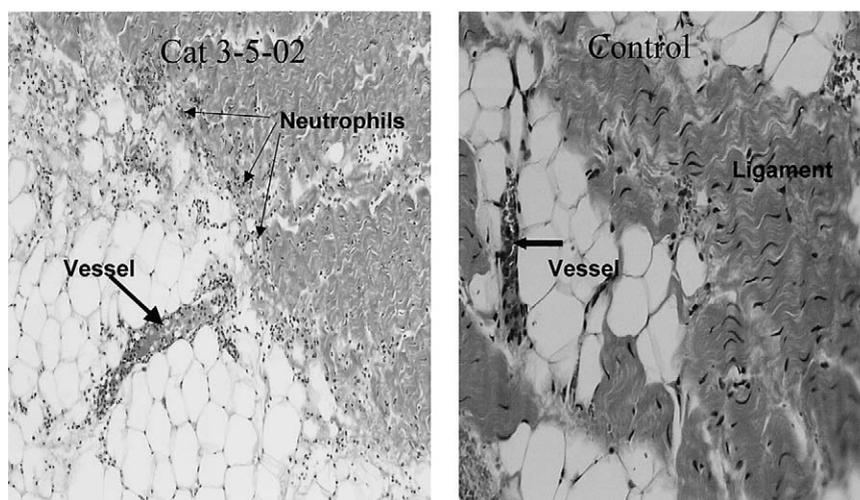

Fig. 9. A typical slide of a supraspinous ligament section after 6 hours rest following 20-minutes static loading is shown on the left. It shows a large number of neutrophils infiltration from a small blood vessel, as well as a substantial number of neutrophils already transported into the tissue, indicating that a full inflammatory process is underway. On the right is a slide from a control preparation which was not subjected to any static or cyclic loading. Only a few spontaneous neutrophils are present.

The various viscoelastic tissues, especially the posterior ligaments, however, will provide most of the forces required for posture and stability in deep flexion. In that case, sustained flexion may initiate the creep and micro-damage in the viscoelastic tissues which in turn trigger spasms and later the initial and morning after hyperexcitabilities.

We believe that the data described in this report provide the biomechanical, electromyographical and histological infrastructure that explain and describe this common low back disorder including its source, symptoms and course over time. This disorder is triggered by sustained lumbar flexion, static or cyclic, which develops micro-damage in the viscoelastic tissues. In turn, the damage triggers spasms in the lumbar muscles during the sustained flexion, and two types of muscular hyperexcitability during rest following the flexion which are shown schematically in Fig. 10. The first hyperexcitability observed in the first hour after work is a compensatory mechanism to the laxity in the lumbar intervertebral joints due to the creep developed in the ligaments, discs, facet capsule, etc. The muscles are triggered into providing the forces necessary to maintain stability and to prevent further damage to the already compromised tissues. Once the creep substantially recovers, the hyperexcitability diminishes. The micro-damage, however, triggers a time dependent metabolic process to repair the damage in the tissue. This inflammatory process is dependent on circulatory transport of

neutrophils to the affected tissues over several hours. The time when the inflammation and its associated muscular hyperexcitability reach their peak seems to be also dependent on the load magnitude. Low loads result in relatively short periods of delayed hyperexcitability as well as mild inflammation. Higher loads may require much longer time of muscular protection as the inflammation is probably severe. The increase in muscular activity during this 'morning after' period is provided to increase joint stiffness, unload the viscoelastic tissues and prevent additional damage while allowing healing.

While this common idiophatic low back disorder is transient, resolving itself within 1–3 days, it may have significant implications in other conditions, including explanation of the physiology and biomechanics of cumulative trauma disorders. The key issue in extending the insight gained from this research into the cumulative trauma disorder is that the creep developed in the viscoelastic tissues did not fully recover after 7 h of rest and that inflamation was set in the viscoelastic tissues. In fact, the displacement pattern during the late recovery period, in the 6–7th hour shows an asymptotic curve with very little slope. It is doubtful if a full recovery could be obtained within the next 12 h. Workers exposed to static or repetitive lumbar flexion are subjected to development of gradually increasing creep in their viscoelastic tissues, and over time, the development of chronic inflamation which is not treatable. This may render the worker as permanently disabled with the severe personal,
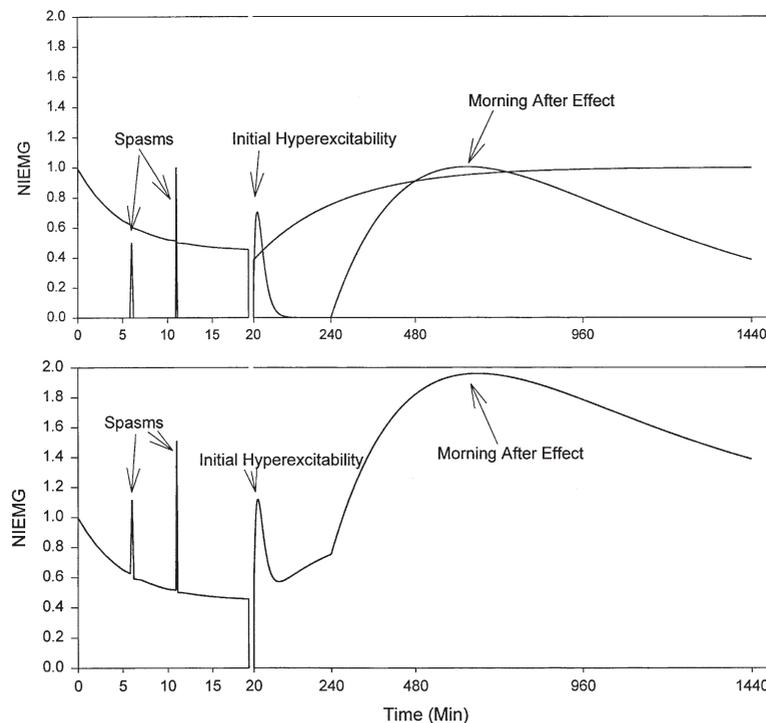

Fig. 10. A schematic of the neuromuscular disorder components is shown in the top whereas the combined effect of the components is shown on the bottom.

psychological and financial implications associated with disability. One preventive measure is the design of an appropriate work - rest schedule which will allow the creep developed in the work session to fully recover before initiating the next work period. Rotating workers within several work duties requiring different physical engagement conditions over one day or from day-to-day may also be of value.

From the medical treatment standpoint, patients presenting this type of idiophatic low back disorder could benefit from anti-inflammatory medication and rest, as that may directly address the cause of the complaint and accelerate the resolution of the disorder. Muscle relaxant or pain medication may treat only the symptoms but not the source of the problem.

Whereas some of the major physiological, histological and biomechanical factors associated with the development of this type of low back disorder were delineated, the impact of several other factors is still unknown. The recovery of performing a repetitive task may have a pronounced impact on the damage inflicted on the viscoelastic tissues. The epidemiological data supports such a hypothesis [16,38,43]. The biomechanical literature further asserts that fast rates of stretch applied to viscoelastic tissue may be associated with permanent damage or even rupture. These issues need to be explored in depth.

Another issue of importance is the optimal work–rest periods that may prevent the development of cumulative disorders. From the data obtained so far, rest periods should be substantially longer than work periods if one wishes to allow sufficient recovery of creep. The exact periods and their ratios compounded with the load magnitude and repetition rate is still unknown.

The data presented above were collected from the feline model, a quadriped. One needs to assert that the same histological, physiological and biomechanical processes are active in the human as well before extrapolation and use in the medical and occupational fields. Work in progress in the Occupational Medicine Research Center and elsewhere points out that the processes described above are indeed the same processes active in humans [3,52]. A large group of young male and female students were subjected to a 10-min period of static lumbar flexion. Electromyographic and video recordings confirmed that spasms were present in 48% of the females and 74% of the males during the static flexion. Flexion–relaxation tests performed before and after the static flexion reveal that the paraspinal muscles were active longer during the flexion and initiated activity earlier during extension, exhibiting a neuromuscular dysfunction. Similar findings were recently observed by Dolan et al. [6] in a similar protocol.

Additional recent work in our Center shows a similar development of a neuromuscular dysfunction in normal human subjects whose anterior cruciate ligament was exposed to static load for 10-min periods [3]. Over 35%

of the subjects exhibited spasms during the static load period and significantly larger maximal voluntary extension and flexion contractions were evident in the quadriceps and hamstrings, respectively. The antagonist co-activation, however, failed to compensate for the increased agonist activity, further compounding the disorder.

Overall, it seems that static or cyclic loads induce the same musculoskeletal disorder in humans; i.e., spasms during the tissue loading and hyperexcitability of muscles after the tissue loading. Ongoing work in our Center also explores the inflammatory process in humans.

Acknowledgement

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