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Neuromuscular disorders associated with static lumbar flexion: a feline model

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Abstract

Static flexion of the lumbar spine with constant load applied to the viscoelastic structures for 20 minutes and for 50 minutes resulted in development of spasms and inhibition in the multifidus muscles (e.g., deep erector spinae) and in creep of the supraspinous ligament in the feline model. The development of spasms and inhibition was not dependent on load magnitude. It is suggested that occupational and sports activities which require prolonged static lumbar flexion within the physiological range can cause a "sprain"-like injury to the ligaments, which in turn reflexively induce spasms and inhibition in some erector spinae muscles. Such disorder may take a long time to recover, in the order of days to weeks, depending on the level of creep developed in the tissues.

Keywords: Spine; Muscle; EMG; Spasm

1. Introduction

Workers engaged in occupational activities which require prolonged static lumbar flexion (e.g., mechanics, carpet and brick layers, farm workers, concrete workers, assembly lines, etc.) report episodes of low back disorders at a rate of up to 10 times that of the general work force [2,3,12–14,20]. A significant number of such complaints are classified as idiopathic low back pain due to the lack of pathological findings (e.g., herniated disc, impingement, etc.) upon examination. While it is suspected that the prolonged static lumbar flexion is the cause of the disorder, its actual physiological mechanism is not known. It was thought that fatigue of the paraspinal muscles during sustained flexion may cause the disorder. Deep flexion, however, is known to be subjected to the flexion–relaxation phenomenon (e.g., deactivation of the muscles half way into full flexion) [6], which renders the fatigue explanation as untenable, since the muscles are not active and therefore would not fatigue.

One possible source for the disorder described above could be from disturbances in the sensory mechanism of the lumbar spine. Sensory receptors consisting of the Golgi, Ruffini, Pacinian and bare endings were shown to exist in the various ligaments of the spine, in the disc and capsule [8]. Mechanical and electrical excitation of the various sensory receptors in the ligaments, disc and capsule were shown to elicit a reflex activation of the paraspinal muscles [10,11,24,26]. Recently, static stretch of constant displacement of the supraspinous ligament was shown to elicit strong spasms in the multifidus muscles superimposed on their reflexive activity in response to the ligamentous stretch and deformation of the disc and capsule [27]. Spasms were long considered as a neuromuscular disorder and are commonly associated with pain or severe discomfort [5,9,16,19,21,22].

It is still unknown if such spasms are elicited as response to the long lasting increase in the ligaments length (e.g., creep) or as response to the loss of the ability to sustain the applied load in the ligament (e.g., tension–relaxation). Since the four types of mechanoreceptors (Golgi, Pacinian, Ruffini and bare endings) found in the spinal viscoelastic tissues are sensitive to elongation and/or to load [4,17,25], it is of interest to assess if neuromuscular disorders could develop as

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response to constant load applied to the spinal viscoelastic tissues.

The objective of this investigation is to revisit the electromyographic response of the lumbar deep erector spinae muscles as response to static constant load (as opposed to the constant displacement employed before by Williams et al. [27]) applied to the supraspinous ligament. It is hypothesized that a static constant load applied to the ligaments also will, over time, elicit spasms in the multifidus muscles. It was also intended to assess if the load magnitude had any impact on the development of spasms in the deep erector spinae muscles.

2. Methods

2.1. Preparation

Eighteen cats (4.78 kg±0.32 kg) were anaesthetized with a single injection of chloralose (60 mg/kg) in a protocol approved by the Institutional Animal Care and Use Committee (IACUC). The skin over the spine was dissected from the thoracic level to the sacral level and reflected laterally, to expose the intact dorso–lumbar fascia. The preparation was placed in a rigid stainless steel frame that allowed the isolation of various lumbar levels via external fixation. Thirteen preparations were used in two experimental groups; seven and six in each group. Five preparations were used in a control group.

2.2. Instrumentation

Six pairs of stainless steel fine wire EMG electrodes, insulated except for a 1 mm exposed tip, were inserted via hypodermic needles into the deep erector spinae (multifidus) muscles of the L-1/2, L-2/3 . . . L-6/7 on the right side, 5–6 mm from the midline. The interelectrode distance of each pair was 3–4 mm. A ground electrode was inserted into the hind limb. Each electrode pair constituted the input to a differential amplifier of 110 dB common mode rejection ratio, a gain of up to 200,000 and a bandpass filter of 6–500 Hz. EMG response from each channel was monitored on oscilloscopes, and stored in the computer with a sampling rate of 1000 Hz. Although intramuscular wire electrodes were used, the low pass filter cut-off frequency was selected to be 500 Hz instead of 1000 Hz in order to minimize the sampling rate to 1000 Hz (e.g., 2×500 Hz=1000 Hz). Since continuous recordings were made over 20 and 50 minutes, sampling at higher rates required a significant amount of storage space which was not available, as well as compromise in processing speed in the analysis phase. Overall, since power spectra analysis was not relevant in this study, the lower cut-off frequency had no significant impact on the data or its analysis.

An "S" shaped stainless steel hook was inserted around the middle part of the L-4/5 supraspinous ligament and connected to the actuator of a Bionix 858 Material Testing System (MTS, Inc., Minnesota) instrumented with a computer controlled loading system. The load cell and displacement outputs of the Bionix 858 were sampled into the computer along with the EMG data.

The lumbar spine was isolated by applying one external fixator to the L-1 posterior spinous process, and a second fixator to the L-7 process, as shown in Fig. 1. The external fixation was not intended to prevent micro or macro motion of the vertebrae, but to limit the elicited flexion to the lumbar spine and prevent interaction of thoracic and sacral/pelvic structures.

2.3. Protocol

The two experimental groups were subjected to the protocol described below, whereas the control group was prepared identically but left undisturbed (e.g., unloaded) for a 50 minutes period while recording EMG from all channels. This was done in order to determine if the dissection performed elicited any of the EMG activity that may be confused as response to the load application in the experimental group.

In the two experimental groups, the stainless steel hook applied to the L-4/5 supraspinous ligament was pulled up by the Bionix 858 system while controlling a ramp-and-hold load of 50 N from a resting position with a 1.0 N preload (applied just before the ramp-and-hold). The load was ramped to 50 N within the first six seconds. The load was applied for a period of 50 minutes in seven preparations, following which it was returned

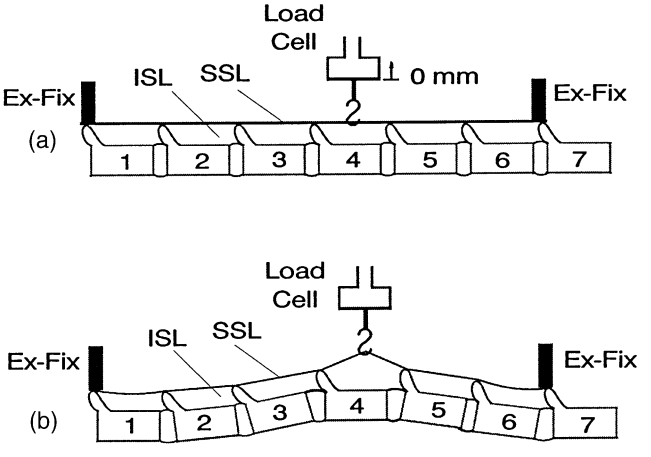

Fig. 1. A schematic representation of the experimental set-up showing the L-1 to L-7 lumbar spine, the supraspinous (SSL) ligament and the intraspinous (ISL) ligament, the "S" shaped hook and the actuator/load cell of the Material Testing System which loaded the L-4/5 SSL into lumbar flexion. The figure also shows the external fixator (ex-fix) which was applied to isolate the lumbar spine and prevent interaction with thoracic and sacral segments.

to the original baseline (e.g., zero load at rest). A 50 N load was calculated and tested to provide mostly axial strains in the L-4/5 supraspinous ligament within its physiological range [18,27], in the initial phase of the loading. The applied load also shifts the adjacent rostral and caudal lumbar vertebrae into moderate flexion such that viscoelastic structures (various ligaments, discs and facet capsules) of several lumbar motion segments were deformed as well. It is expected that the overall response is, therefore, due to excitation of mechanoreceptors in the viscoelastic structures of several levels [24].

Using two short hypodermic needles inserted into the spinous processes of L-4 and L-5, the length of the supraspinous ligament of that segment was measured with calipers while the 1.0 N preload was applied just before the onset of the 50 minutes flexion session. Immediately after the step and hold displacement was returned to zero, the load was reset to 1.0 N (in order to offset the creep (laxity) developed in the ligament during the 50 minutes sessions) while recording the vertical displacement of the load cell required to elicit the 1.0 N load. The L-4/5 supraspinous ligament length was also remeasured [27] (see Appendix A). The measurements were used to estimate the final creep (residual axial strain) in the ligament developed by the loading period.

In six preparations different magnitudes of load, 20 (N=2), 30 (N=2), 50 (N=1) and 70 (N=1) N were applied for a 20 minutes period in order to assess if the load magnitude had a direct impact on the development of spasms.

EMG from the six multifidus muscles, displacement and load were recorded continuously over the loading period.

A gauze pad soaked with saline was applied over the incision during the trials in order to prevent the exposed tissue from drying.

2.4. Analysis

In the first experimental group (N=7), two-second-wide windows of EMG from each of the six spinal levels were sampled immediately at the beginning of the loading period, and every 20 seconds thereafter for the 50 minutes loading period. Each EMG sample was integrated over the two second window, and normalized with respect to the first sample. The normalized integrated EMG (NIEMG) of all the preparations at the respective window were pooled, and the mean (±SD) was calculated and plotted on a NIEMG vs time plot for each of the muscles of the six levels.

Similarly, the displacement recorded just at the beginning of the loading period was used for normalization of the displacements sampled subsequently. Normalized displacements of the respective window of all preparations were pooled, and the mean (±SD) was calculated and plotted as a normalized displacement vs time plot.

By using the measurements of the supraspinous ligament length at 1.0 N preload before and after the 50 minutes displacement was applied, and the vertical displacement of the load cell required to elicit 1.0 N load at the end of the 50 minutes trial, the final value of the creep in the ligament was calculated [27] (see Appendix A).

A model was developed to describe the pooled NIEMG in the form of an exponential decay with a non-zero asymptote of the following form:

$$\mathrm{NIEMG} = A\exp(-at) + R$$

where A=exponential component initial amplitude; a=exponential component decay rate constant; R=steady state NIEMG amplitude; t=time.

The normalized actuator displacement (D) was modeled as an exponential to maximum. The equation is of the following form:

$$D = B(1-\exp(-bt)) + 1$$

where D=normalized actuator vertical displacement; B=steady state strain; b=rate constant; t=time.

The equation parameters (A, a, R for the NIEMG, B, b for the displacement) were fitted using the Marquardt–Levenberg non-linear regression algorithm. In the fitting process for the NIEMG, the following constraint was used:

$$A + R = 1$$

This constraint forced the model to attain a value of 1 at time 0.

3. Results

Figure 2 provides typical recordings of EMG, displacement and load during the 50 minutes of continuous load application from three different preparations from the experimental group (N=7) subjected to 50 minutes static flexion at constant load. The applied 50 N load to the supraspinous ligament at the L-4/5 level is shown in the bottom trace. The trace above it shows the displacement of the MTS actuator as the lumbar viscoelastic tissues (ligaments, discs and capsules) underwent creep while the load was kept constant at 50 N. The six traces above are the recordings of EMG from the L-1/2 to the L-6/7 levels.

In Fig. 2, the EMG traces show initial large response with a general exponential decay with time. In the EMG trace of the L-1/2 in the left column, a burst of large amplitude EMG spasms is present in the first minute, lasting several minutes. Similar bursts of EMG are also present between the 25th minute and the 38th minute.

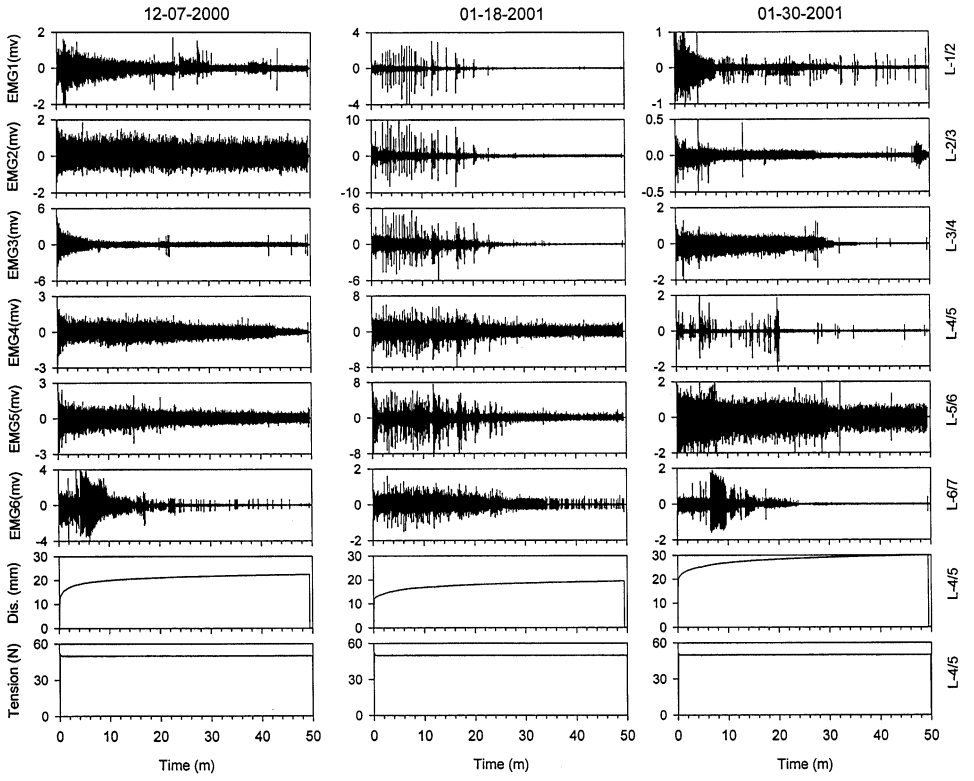

Fig. 2. Typical recordings of EMG, vertical displacement and constant load during the 50 minutes test from three different preparations loaded to 50 N. Note the general trend of exponential decrease in EMG over time with spasms superimposed on the exponential behavior. The spasms were of different intensity and frequency and appeared at different times in each of the three preparations.

Spasms of large amplitude are also clearly visible in the L-6/7 trace, initiating in the 5th minute and lasting for 4–5 minutes. The EMG trace of the L-3/4 level also displays a large discharge at the 22nd minute.

Spasms are also evident in all the six traces of the middle column throughout the first 25 minutes. In this preparation the spasms begin one minute after the application of the load and remain superimposed on the EMG discharge for the 25 minutes thereafter. Most of the spasms are expressed as large amplitude compound action potentials superimposed on the EMG, and forming several waves between the 10th to the 25th minutes.

In the right column, an abrupt and intense discharge appears in the L-6/7 level at the 7th minute lasting for three minutes and then followed by three smaller waves. In the L-2/3 level, the gradually decreasing EMG is interrupted by several large bursts in the initial 15 minutes, and an intense low amplitude wave between the 47th and 49th minutes. Large sporadic action potentials are seen throughout the 50 minutes period in L-1/2 and L-4/5. A sharp inhibition is present after the 32nd minute in L-3/4, and after the 22nd minute in L-6/7.

Another important feature in the typical displays of Fig. 2 is the gradual increase in the displacement due to the creep developed by the constant load. The initial displacement upon application of a 50 N load was 13–14 mm in two of the three specimens and 21 mm in the third one and reaching 22 mm to 30 mm at the end of the 50 minutes trial.

Figure 3 presents the pooled and normalized EMG (NIEMG) and displacement data of the seven experimental preparations during the loading period. The exponentially decreasing trend of the EMG is evident despite the random interruption by spasms throughout the 50 minutes loading period. The testing at 1 N load before and after the 50 minutes loading period resulted in a mean axial creep developed in the L-4/5 supraspinous ligament amounting to 18% (i.e., 18% larger than its original length).

Spasms were evident in each of the six preparations in which the load applied to the lumbar spine varied. Figure 4 displays sample recordings from the preparations loaded at 20, 30, 50 and 70 N. The left column presents the recordings from one of the two preparations subjected to the lowest test load of 20 N, which is just above the trigger threshold of the reflex [24]. The initial EMG activity in all channels decreases exponentially to a baseline, and then at the ninth minute large spasms are seen in L-1/2, L-4/5 and L-5/6, lasting to the end of the period. Small discharge is also present in the L-3/4 level. In the second column from the left large amplitude spasms erupted in L-1/2 and L-2/3 after the seventh minute while simultaneously the EMG was inhibited in

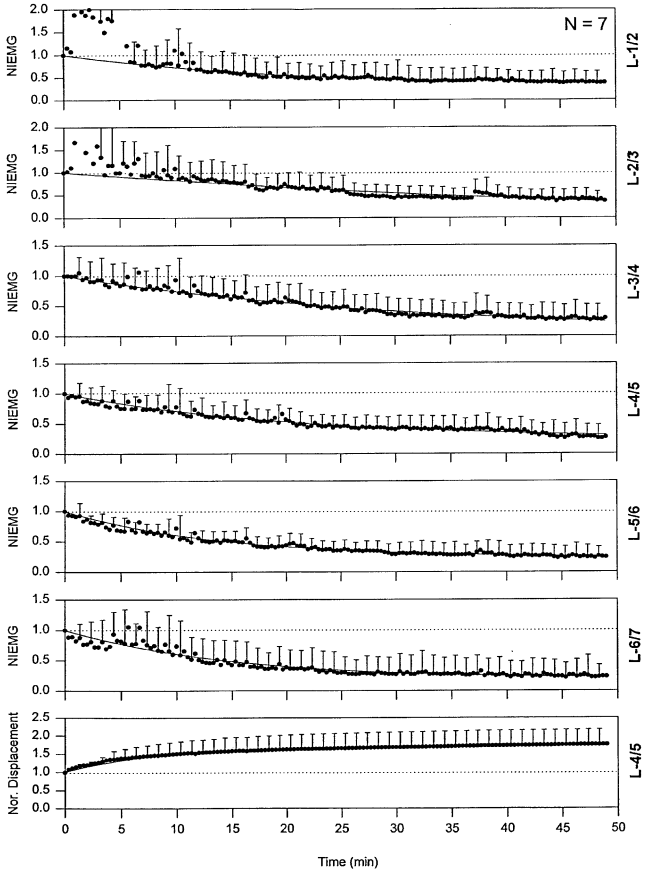

Fig. 3. The pooled mean (+SD) normalized integrated EMG (NIEMG) and the normalized displacement (bottom trace) of the 7 preparations tested at 50 N constant load over 50 minutes. The models developed for the EMG from each lumbar level and for the displacement are superimposed on the pooled data. Note that the large variability introduced by the spasms are present rather obviously in the NIEMG traces of L-1/2, L-2/3 and L-6/7 and to a lesser extent in the other levels.

the L-3/4 and L-4/5 levels. Spasms were also seen in L-5/6 and L-6/7 starting at the third minute.

In the third column from the left, large amplitude spasms were recorded in L-1/2 and L-2/3 at the seventh minute and in L-3/4 starting at the 14th minute. Spasms were present in L-5/6 in the 16th minute while in L-6/7 random discharge appeared throughout the 20 minutes. In L-4/5 no spasms were evident.

In the right column large amplitude compound action potentials were superimposed on the reflexive activity of the muscles of all levels throughout the loading period with two specific waves at the L-6/7 level between the 12th and 18th minutes. A distinct wave was also seen in the L-1/2 level starting at the end of the first minute and lasting until the fourth minute.

EMG recordings from the control group did not show activity above the normal baseline EMG throughout the 50 minutes of observation, confirming that the EMG activity seen in the experimental group was due to the load applied and not to the dissection or any other uncontrolled factor.

The models developed for the pooled NIEMG and displacement shown in Fig. 3 had the anticipated form described in the Methods section.

The equation parameters (*A*, *a*, *R* for the NIEMG, *B*, *b* for the displacement) were fitted by the Marquardt–Levenberg non-linear regression algorithm. The resulting fit parameters with their corresponding correlation coefficients are shown in Table 1. In general, residual levels in the range from 3 to 33% were found with the exception of L-1/2, which according to the model would settle to 0 if given enough time. Correlation coefficients were greater than 90% with the exception of L-1/2 and L-2/3. This is most likely due to the fact that in these cases, spasms occurring within the first five minutes drew the means above unity, increasing the model's error. The model, being constrained to remain below unity, was unable to account for the spasms, particularly when they occurred early and in channels with low initial EMG levels.

4. Discussion

The most important findings of this investigation were that static constant load applied to the lumbar spine via the supraspinous ligament results in spasms of the multifidi (deep erector spinae) muscles although the ligament was stretched well below its physiological range limit. The spasms were also induced while superimposed on the initial reflexive EMG activation level of the deep erector spinae muscles which tended to decrease with time while subjected to the static load. The amplitude, duration and timing of the erratic EMG activity as well as the lumbar levels in which it appeared were unpredictable and random which by definition fits the description of spasms. Spasms were evident regardless of the magnitude of the load applied to the lumbar spine, including loads just at the trigger threshold of the reflex.

Spasms and elevated activity of the lumbar paraspinal muscles are common in patients with low back pain. The spasms and elevated muscular activity have also been confirmed electromyographically [5,7,9,16,21,22]. In some of the cases studied by the above investigations the cause of the low back pain was confirmed (such as herniated disc, impingement), while others were idiopathic. Furthermore, Pedersen et al. [19] have shown that crushing of the supraspinous ligament in the feline results in recording of spasms in the gluteus muscles, experimentally establishing the relationships between tissue damage and spasms. Correlating all the fragments of information above suggests that a distinct chain of events consisting of viscoelastic tissue damage–pain–spasm exists.

It is important to note that the supraspinous ligament

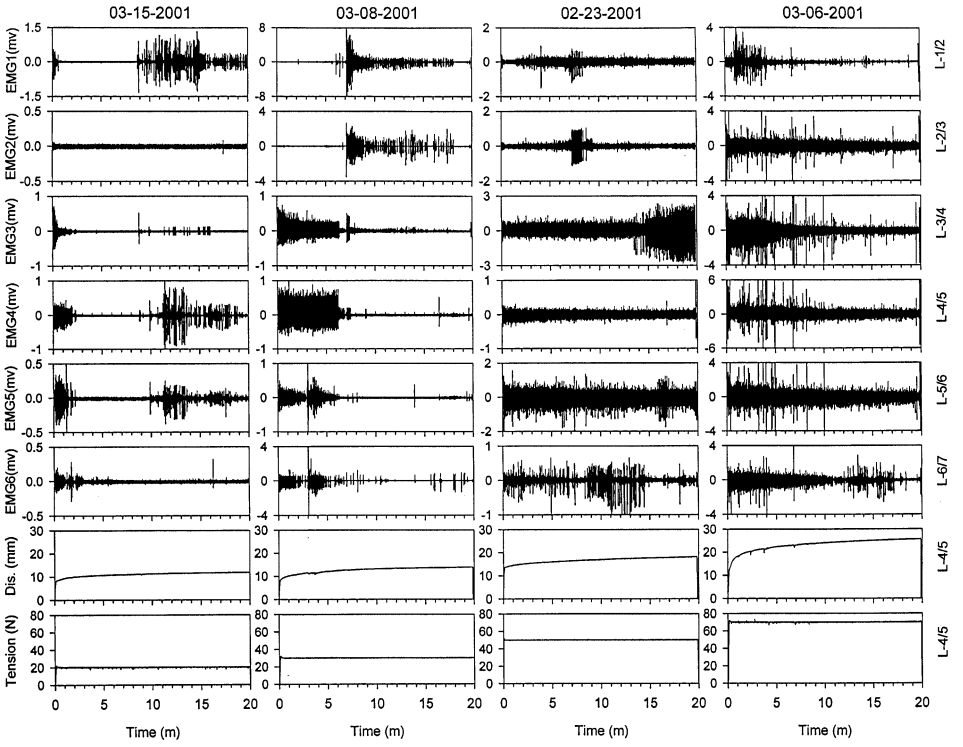

Fig. 4. Typical recordings of EMG, displacement and load from separate preparations. The preparations on the left were loaded to 20 N while the following three to the right were loaded to 30 N, 50 N and 70 N, respectively. Exponential decrease of EMG and spasms were evident regardless of the load magnitude.

Table 1
Model parameters for NIEMG and displacement

Level	*A*	*a*	*R*	R^2
L-1/2	0.6639	0.0544	0.3321	0.3471
L-2/3	1	0.0217	0.00	0.6269
L-3/4	0.9629	0.0319	0.0371	0.9621
L-4/5	0.7573	0.0488	0.2427	0.9581
L-5/6	0.7675	0.0723	0.2325	0.9658
L-6/7	0.8128	0.0627	0.1872	0.9154
Displacement				
	B	*b*	–	R^2
Displacement	0.7173	0.1173	–	0.9653

was loaded such that the resulting strains (i.e., stretch) were within its physiological limits [18,27]. Both investigations found that the physiological strain in spinal ligaments of human and feline specimens are much larger than that of ligaments in the extremities. Physiological range limits of 25–40% strain were found in the spinal ligaments as compared to 6–7% in ligaments of the extremities. Therefore, the appearance of spasms suggests that damage to the viscoelastic tissues occurred even if the strain is within the tissues' physiological range. While this may explain many cases of low back pain complaints which are not confirmed with observations of any pathology using standard diagnostic techniques, it still requires an explanation of what damage, if any, was inflicted on the tissues. The data confirms that at the end of 50 minutes flexion, the ligament stretched to 118% of its initial resting length and that this residual elongation (known as creep) did not immediately recover its resting length upon removal of the load (i.e., the spine was completely at rest, but the ligament was loose when 1 N was applied). Does the neurology consider the onset of long lasting stretch of the viscoelastic tissues as damage? In the ligaments of the extremities, long lasting excessive strain is designated as a "sprain", is known to last for at least several days, and is often accompanied by pain and muscle spasms.

Another issue revolves around the rate at which the ligament was stretched. Ligaments are known to stretch to significant lengths without showing damage if the stretch is applied slowly. Fast rates of stretch, however, are not tolerated well. Ligaments and other viscoelastic tissues develop extremely large loads when stretched at fast rates and often rupture at lengths not much above their resting length. In this investigation, the ligament was initially stretched as the full load of 50 N was applied over six seconds resulting in a mild rate of stretch in order to avoid damage. It is thought unlikely that the initial rate of elongation was the cause of the damage and spasms.

The magnitude of the load applied to the lumbar spine did not have an impact on the results. Spasms appeared superimposed on the reflexive EMG discharge at random in every preparation including the two which were subjected to an extremely low load of 20 N. This part of the study suggests that a factor other than the load magnitude is responsible for the spasms.

The length of time the stretch was maintained is evidently the prominent factor in causing the creep in the ligament and the consequent spasms. In our previous work [23] we applied cyclic stretch sessions to the same ligament for up to three hours without observing any signs of spasms. The cyclic frequency was 0.25 Hz, allowing two seconds for gradual stretch and two seconds for relaxation. Therefore, the continuous static stretch over time applied in this investigation causes ongoing creep in the ligament without allowing it any intermittent periods to recoil to the resting position and recover. As the creep increases with time the ability to instantaneously recoil is lost and with that the functional properties of the tissue. That, perhaps, constitutes the damage to the tissue and the trigger of spasms. It seems, then, that the static ongoing strain in ligaments is the primary factor that triggers the chain of creep–pain–spasms.

The EMG recorded from the different levels of the lumbar spine demonstrate a gradual decrease with time (when the spasms are disregarded) as shown in Fig. 3. At the same time the viscoelastic structures (discs, ligaments and capsules) demonstrated the development of creep (e.g., stretching) as is also shown in the bottom trace of Fig. 3. It is important to remember that the load applied to the spine via the supraspinous ligament, however, was kept constant. One can conclude, therefore, that the tension developed in the viscoelastic structures may be one stimulus that elicits the reflexive activity of the deep erector spinae muscles. It is also clear that some type of accommodation takes place which causes decrease in the EMG discharge over time despite the applied constant load. Overall, the laxity developed in the spine together with the reduced contribution of muscular forces add up to reduced stiffness and therefore reduced stability and increased exposure to additional injury.

The development of creep in the spine when human subjects performed static flexion was described before [1,15]. Adams et al. [1] also noted that the musculature failed to compensate for the loss in stiffness, confirming that the musculature may not be triggered effectively after a long period of static load application.

The loss in EMG activity as recorded from the muscles over 50 minutes was not as drastic as was seen after the same period of cyclic loading or static loading with constant displacement [23,27]. The EMG activity at the end of the 50 minutes period ranged from 20% to 37% of the initial discharge at the beginning of the 50 minutes. In the cyclic or constant displacement static loading conditions, the terminal EMG discharge was mostly near 5% of the initial discharge. The fact that the stimulus applied continued to maintain constant load suggests that the reflexive EMG discharge is more sensitive to the tension in the viscoelastic structures than to their elongation. This issue was addressed by Solomonow et al. [25] in a separate study when cyclic loads were applied, resulting in sensitivity to both tension and elongation stimuli with increased sensitivity to tension.

While large amplitude spasms were abundant in the recordings made from all the experimental preparations, cases of EMG inhibition were also observed. In Fig. 4, the first and second columns provide the most pronounced examples of the EMG inhibition. In the second column, the EMG discharge from the L-3/4 and L-4/5 is sharply inhibited in the seventh minute and at the same time a very strong EMG discharge is initiated in the L-1/2 and L-2/3. In the first column, the EMG in L-4/5 and L-5/6 is inhibited on the third minute, remains inhibited for eight minutes and then spasms are triggered. It seems that the spinal neurological reflexive network is attempting to decrease and increase muscular activity at different levels in a search for a combination that the interplay of muscular forces will minimize the damage inflicted by the viscoelastic creep. Therefore, elevated muscular activity is not the only disorder associated with static flexion, but alternating muscular inhibition is also an important component of this phenomena.

The reflexive EMG activity elicited by the static flexion was most likely elicited by the large mechanoreceptors found in the ligaments, discs and capsules. Receptors such as Golgi, Pacinians and Ruffini organs are known to signal mechanical events such as tension and deformation of the tissues in which they are embedded. The spasms, however, are most likely triggered by the bare nerve endings which are also found in the same spinal viscoelastic tissues. Bare nerve endings are established as receptors which monitor tissue injury and trigger responses such as pain and probably its associated spasms. It is, therefore, conceivable that two separate sensory feedback mechanisms are active while working in synergy with each other to protect the spine and its important viscoelastic tissues from instability and injury.

The reader should note that the data presented in this report was obtained from the feline and that some consideration should be taken before the conclusions are applied to the human. Inherently, differences exist between the human and the feline, the most prominent of which is the fact that they are bipeds and quadrupeds, respectively. Consequently, the human is subjected to the gravity vector which is parallel to the spine whereas the feline's spine is perpendicular to the gravity vector. Despite the differences in the orientation and effect of gravity on muscular responses, the neuromuscular sys-

tem in the human and feline (both are mammalians) are similar. Nerves, muscles, ligaments, bones, spinal cord and many brain functions and structures including reflexes are established to be similar in the neurophysiological literature. There are, however, differences in size, structure and function that should be clearly understood when one attempts to interpret the data and predict conclusions relative to human responses. The vertebrae, ligaments, discs, etc., are much smaller in the feline when compared to human. Nevertheless, when one considers the overall weights and loads in proportion to size, the biomechanical/physiological functions scale in proportion such that the overall observed phenomena are not different. Results from data collected from the feline could apply to humans with an appropriate scaling for size differences.

Furthermore, when considering the differences in the orientation to the gravity vector, the spinal ligaments in the human are much larger. The supraspinous ligament, for example, is thicker in the human not only due to size scaling, but also due to evolutional hypertrophy as response to additional resistive stiffness to gravity.

A feline model also presents unique advantages in identifying isolated fundamental stimulus and its response while being more physiologically representative, since it is possible to eliminate secondary and tertiary influences which are always present in research using human subjects. Factors such as visual, auditory and psychological reactions in human subjects normally have strong artifactual interference that prevent the understanding of an isolated stimulus–response phenomenon. An anaesthetized feline specimen allows the advantages of studying such basic biomechanical/physiological phenomena while performing invasive surgery which is not allowed in human subjects.

Overall, when combining the basic biomechanical/physiological mechanisms of function or disorder developed from animal models with performance data from humans in the laboratory or workplace and with epidemiologic data, one can gain a significant insight into many aspects of disorder development and its prevention.

5. Conclusion

From the data resulting from this study, the following conclusions could be made:

1. Static constant load developed in the lumbar viscoelastic tissues first initiates a reflex activation of the deep erector spinae muscles such that joint stiffness and its stability is maintained.
2. The activity of the muscles diminishes with time despite the fact that constant load was maintained.
3. Static flexion of the spine triggers a neuromuscular disorder consisting of spasms and alternating muscular inhibition. The trigger and inhibition of such muscular activity is random and unpredictable throughout the flexion period.
4. The development of the muscular disorder is independent of the load applied to the tissues, and seems to be dependent on the duration the static load is applied.

Overall, static lumbar flexion could develop sufficient creep in the lumbar viscoelastic structures to trigger a muscular disorder which is probably associated with pain and discomfort. Static loading of the spine, therefore, should be avoided or minimized as much as possible in order to avoid occupationally related problems.

Acknowledgements

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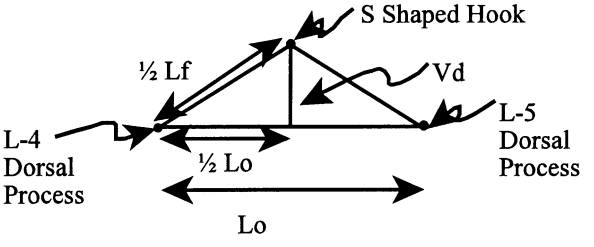
Appendix A

Estimation of the residual strain in the L-4/5 supraspinous ligament at the end of the 50 minutes lumbar flexion:



where *Lo*=length of the supraspinous ligament at 1.0 N before the 50 minutes of loading; *Lf*=length of the supraspinous ligament at 1.0 N after the 50 minutes of loading; *Vd*=vertical displacement of the load cell required to elicit 1.0 N load immediately after the 50 minutes of loading.

$$Lf = 2\sqrt{\left(\frac{1}{2}Lo\right)^2 + Vd^2} \quad (1)$$

and:

$$\text{Residual strain} = \frac{Lf - Lo}{Lo} \times 100\% \quad (2)$$

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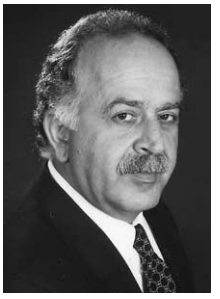

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