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Multifidus Spasms Elicited by Prolonged Lumbar Flexion

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Study Design. The electromyogram of the L1–L7 multifidus muscles of the *in vivo* cat were recorded while applying a prolonged steady displacement to the lumbar spine through the L4–L5 supraspinous ligament, simulating a moderate anterior flexion.

Objective. To demonstrate that tension-relaxation and laxity of the viscoelastic structures (ligaments, discs, and capsules) induced by prolonged static flexion of the spine results in loss of reflexive muscular stabilizing activity and in muscular disorders that may lead to or are associated with low back pain.

Summary of Background. Epidemiologic data show that prolonged loading of the spine, such as in some occupational activities, can cause low back pain and muscle spasms. Direct experimental evidence linking prolonged loading to a decrease in spinal stability, low back pain, and muscle spasms was not found. It was hypothesized, however, that mechanoreceptors in the viscoelastic structures, when strained, reflexively activate the multifidus muscles to maintain intervertebral stability; that the reflexive muscular activity decreases with stress-relaxation and laxity in the viscoelastic structures; and that when severe strain and possible damage of the viscoelastic structures occurs with time, nociceptive receptors elicit spasms in the musculature and possible pain.

Methods. The lumbar spine of seven *in vivo* cat preparations was displaced through the L4–L5 supraspinous ligament into moderate flexion that was steadily maintained for 50 minutes while intramuscular electromyograms were recorded from each of the multifidus muscles of L1–L2 through L6–L7. Load and electromyogram were continuously monitored and recorded. Five additional preparations were used as controls, in which dissection and recordings were identical, but the lumbar flexion was excluded.

Results. Prolonged flexion of the lumbar spine resulted in initial reflexive electromyogram from the multifidus muscles that decreased to approximately 5% of its initial value as tension-relaxation began in the viscoelastic structures within the first 3 minutes, after which, random and unpredictable electromyogram discharges (*i.e.*, spasms) of high amplitude were recorded from different levels. In some preparations the spasms were present in L1–L4, and in others in all the levels. In other preparations the spasms were recorded only at L5 and L6. The onset of the spasms was also unpredictable, because they were initiated in some cases within 2–3 minutes after the spine was loaded. In other cases, the spasms were observed anytime during the test period and up to 20 minutes after the load was removed. Spasms were also observed in the spinalis and longissimus muscles.

Conclusions. Prolonged flexion of the lumbar spine results in tension-relaxation and laxity of its viscoelastic structures, loss of reflexive muscular activity within 3 minutes and electromyogram spasms in the multifidus and other posterior muscles. [Key words: electromyogram, ligament, low back pain, lumbar, multifidus, spasms spine] **Spine 2000;25:2916–2924**

It is well established that the musculature associated with the lumbar spine is the primary structure responsible for its stability, whereas the passive viscoelastic structures (ligaments, discs, and capsules) function as secondary stabilizers.[5,7,9,16,21] The musculature and the viscoelastic tissues of the spine, however, function synergistically, so that the desired movement is accomplished while the stability of the spine is preserved.[21] Recent evidence has shown that a spinal reflex arc exists from mechanoreceptors in the ligaments, discs, and facet capsules to the lumbar multifidus and longissimus muscles in humans[28] and in feline[28,29] and porcine[13,14] models. Strain of the lumbar viscoelastic structures was shown to excite the mechanoreceptors within and reflexively contract the multifidus muscles of the distracted motion segment. Partial activation of the multifidus muscles of two to three levels above and below was also observed. Such muscular forces provide sufficient stiffness to prevent excessive displacement of several vertebrae relative to each other and the consequent possible injury.[28]

More recently, Solomonow et al[27] and Gedalia et al[6] have shown that tension-relaxation and the associated laxity induced in the viscoelastic structures due to cyclic loading of the lumbar spine desensitize the mechanoreceptors within. The resultant biexponential decrease of reflexive muscular activity of the multifidus leaves the spine without muscular protection against excessive intervertebral displacement.

Adams et al[1] showed that creep in the viscoelastic structures was also present when the spine was subjected to steady load over time, as would be expected during prolonged anterior flexion while gardening or bricklaying, for example. They also observed that in human subjects, the musculature did not compensate for the lost stiffness (*e.g.*, laxity) in the spine. The exact correlation between the load applied to the spine, the laxity induced,

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and the changes in the level of muscular activity over time, however, is not known.

It is the objective of this research to evaluate the pattern and time course of reflexive contraction of the multifidus elicited by prolonged static flexion of the lumbar spine of an *in vivo* feline preparation. It is anticipated that such data can shed light on the mechanism of low back pain development in workers engaged in occupational activities requiring the prolonged assumption of a given posture.

■ Methods

Preparation. Twelve cats (4.48 ± 0.53 kg) were anesthetized with a single injection of chloralose (60 mg/kg) in a protocol approved by the Institutional Animal Care and Use Committee. The skin over the spine was dissected from the thoracic level to the sacral level and reflected laterally, to expose the intact dorsolumbar fascia. The preparation was placed in a rigid stainless steel frame that allowed the isolation of various lumbar levels through external fixation. Seven preparations were used as the experimental group, and five preparations were used as the control group.

Instrumentation. Six pairs of stainless steel fine wire electromyogram electrodes, insulated except for a 1-mm exposed tip, were inserted through hypodermic needles into the multifidus muscles of L1–L2 and L2–L3 through L6–L7 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 hindlimb. 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 band-pass filter of 6–500 Hz. Electromyogram response from each channel was monitored on oscilloscopes and stored in a computer with a sampling rate of 1000 Hz.

An S-shaped stainless steel hook was inserted around the middle part of the L4–L5 supraspinous ligament and connected to the vertical armature of a materials testing system (Bionix 858; MTS, Inc., Minneapolis, MN) instrumented with a computer-controlled loading system. The load cell output of the system was sampled into the computer along with the electromyogram data.

The lumbar spine was isolated by applying one external fixator to the L1 posterior spinous process, and a second fixator to the L7 process, as shown in Figure 1. The external fixation was not intended to prevent micro- or macromotion of the vertebrae but to limit the elicited flexion to the lumbar spine and prevent interaction of thoracic and sacral–pelvic structures.

Protocol. The experimental group was subjected to the protocol to be described, whereas the control group was prepared identically but left undisturbed (*e.g.*, unloaded) for a 50-minute period while electromyograms were recorded from all channels to determine whether the dissection performed elicited any electromyogram activity that could be confused with a response to the displacement application in the experimental group.

In the experimental group, the stainless steel hook applied to the L4–L5 supraspinous ligament was pulled up by the materials testing system system, while a step-and-hold displacement of 12–15 mm (according to the specimen size) was controlled from a resting position with a 0.5-N preload applied just

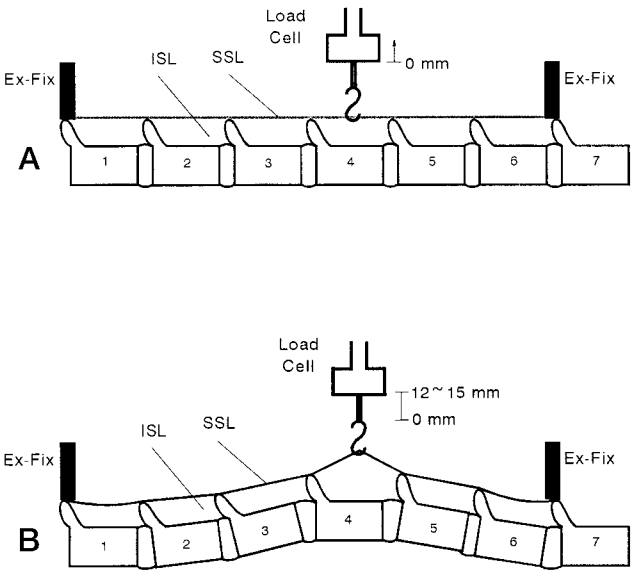

Figure 1. The feline spine, in which the whole lumbar segment was isolated by external fixators at the L1 and L7 processes. **A,** Resting position in which zero displacement was at a preload of 0.5 N. **B,** Condition at which a moderate anterior flexion is simulated by displacing the L4–L5 segment through its supraspinal ligament is shown. This condition was held steady for 50 minutes while recording the load applied and electromyogram in the lumbar multifidus.

before the step and hold. The 12–15-mm displacement was calculated and tested to provide mostly axial strains in the supraspinous ligament within its physiologic range[20] (Appendix A) in the initial phase of the displacement (0–5 mm). Additional displacement (5–15 mm) shifted the adjacent lumbar vertebrae into moderate flexion, so that viscoelastic structures (various ligaments, discs, and facet capsules) of several lumbar motion segments were deformed as well.

To confirm that several lumbar vertebrae and their associated viscoelastic structures were indeed deformed by the applied displacement, one lateral radiograph was taken in the resting state and a second one at full displacement. The displacement was applied for 50 minutes, after which it was returned to the original baseline (*e.g.*, zero displacement at rest).

By using two short hypodermic needles inserted into the spinous processes of L4 and L5 (See Appendix A), the authors measured the length of the supraspinous ligament of that segment with calipers while the 0.5 N preload was applied just before the beginning of a 50-minute flexion session, immediately after the flexion was applied, and just before the 50 minutes of flexion ended. Immediately after the step-and-hold displacement was returned to zero, the load was reset to 0.5 N (to offset the laxity developed in the ligament during the 50 minutes session) while the vertical displacement of the load cell required to elicit the 0.5-N load and the L4–L5 length was recorded. This measurement was used to estimate the residual axial strain in the ligament (Appendix B). Electromyograms from the six multifidus muscles and load were recorded continuously during a 50-minute loading period.

A gauze pad soaked with saline was applied over the incision during the 50-minute trial, to prevent the exposed tissue from drying.

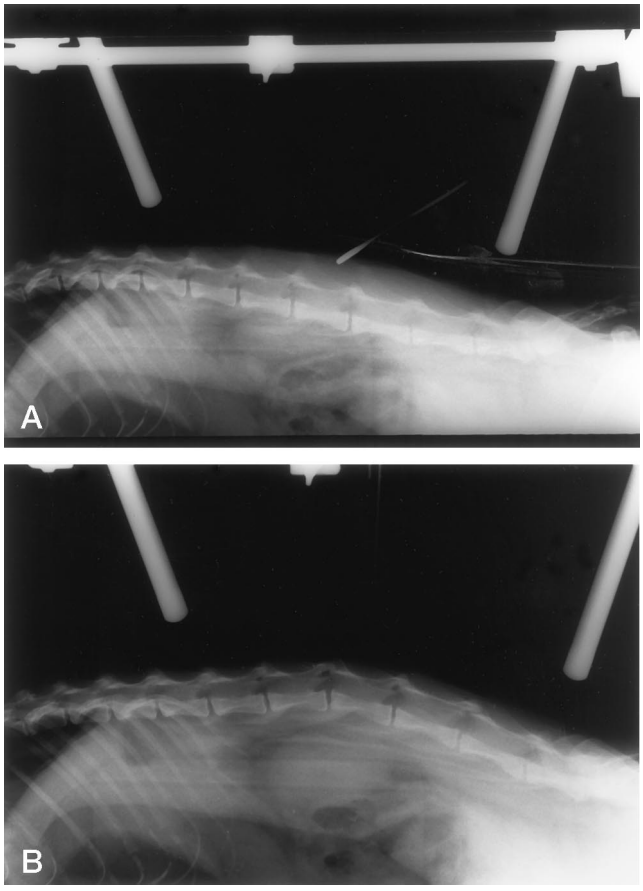

Figure 2. Lateral radiographs of the lumbar spine are shown in the prone cat at rest (**A**) and in the same position after the displacement was applied to the L4–L5 supraspinal ligament demonstrating the moderate flexion induced (**B**).

Analysis. Two-second windows of electromyogram and load applied to the spine were sampled immediately at the beginning of the displacement period, and every 20 seconds thereafter for the first 5 minutes. Each electromyogram sample was integrated over the 2-second window and normalized in relation to the first sample. The normalized integrated electromyograms (NIEMGs) of all the preparations at the respective window were pooled, and the mean (±SD) was calculated and plotted on a plot of NIEMG *versus* time for each of the multifidus muscles of the six levels.

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

By using the measurements of the supraspinous ligament length at a 0.5-N preload before and after the 50-minute displacement was applied and the vertical displacement of the load cell necessary to elicit 0.5-N load at the end of the 50-minute trial, the residual axial strain in the ligament was calculated (Appendix B).

■ Results

Electromyogram recordings from the control group did not show activity above the normal baseline throughout the 50 minutes of observation. Figure 2, A and B, shows the radiographs taken at rest and at full displacement of the lumbar spine, respectively, confirming that moderate flexion was induced in the lumbar levels and that their associated viscoelastic structures were deformed.

Figure 3, A, B, and C, provides the electromyogram and load recordings from three[3] different preparations during the 50 minutes of step-and-hold displacement. The recorded data demonstrate that electromyogram activity was present immediately on application of lumbar flexion and that the electromyogram gradually decreased in the first 3 minutes as tension–relaxation began in the viscoelastic structures. In each of the preparations, randomly appearing electromyogram activity (*e.g.*, spasms) was recorded sometime after the initial electromyogram decayed to a steady-state level. In two of the seven preparations, these electromyogram spasms appeared primarily in the L1–L2, L2–L3, L3–L4 multifidus, in two preparations in the L5–L6 and L6–L7 multifidus, and in three preparations in the multifidus of all levels. In one of the preparations, shown in Figure 3C, electromyogram spasms were first seen in L1–L2 and L2–L3 and later only in the L5–L6 and L6–L7.

In some preparations the electromyogram spasms appeared in two or three distinct waves. For example in Figure 3A, spasms in L1–L2 and L2–L3 appeared first within 2 minutes of displacing the spine, persisted for 2–3 minutes and then diminished. A second wave of electromyogram spasms appeared on the 10th minute, persisted for 4–5 minutes, and then diminished. A powerful third wave appeared on the 27th minute and persisted for 12–15 minutes before diminishing to low-level activity.

The electromyogram spasms in some preparations were of a high-amplitude slow-firing appearance, and in other preparations of a high-amplitude, high-frequency, intense-firing appearance. The timing of the appearance of the electromyogram spasms also varied widely, sometimes triggered within 1–2 minutes of applications of the step and hold displacement, and sometimes triggered 15–30 minutes later.

Although the electromyogram spasms were present in each preparation, the lumbar levels in which they were recorded, the amplitude, and the timing were random from preparation to preparation, so that any type of analysis to establish a predictable pattern was impossible.

It was common to see spasms in the multifidus and visual twitches in the spinalis and longissimus muscles of different lumbar levels after the displacement was removed at the end of the 50-minute trial. Such electromyogram activity was present for as long as 20 minutes after the displacement was removed.

Figure 4 provides the mean (±SD) of the NIEMG from each of the multifidus of the six lumbar levels and the mean (±SD) load of the seven preparations for the first 5 minutes of the loading period. The mean load demonstrated the tension-relaxation that followed an exponential decrease, diminishing to 50% of its original value at the third minute and decreased slowly thereaf-

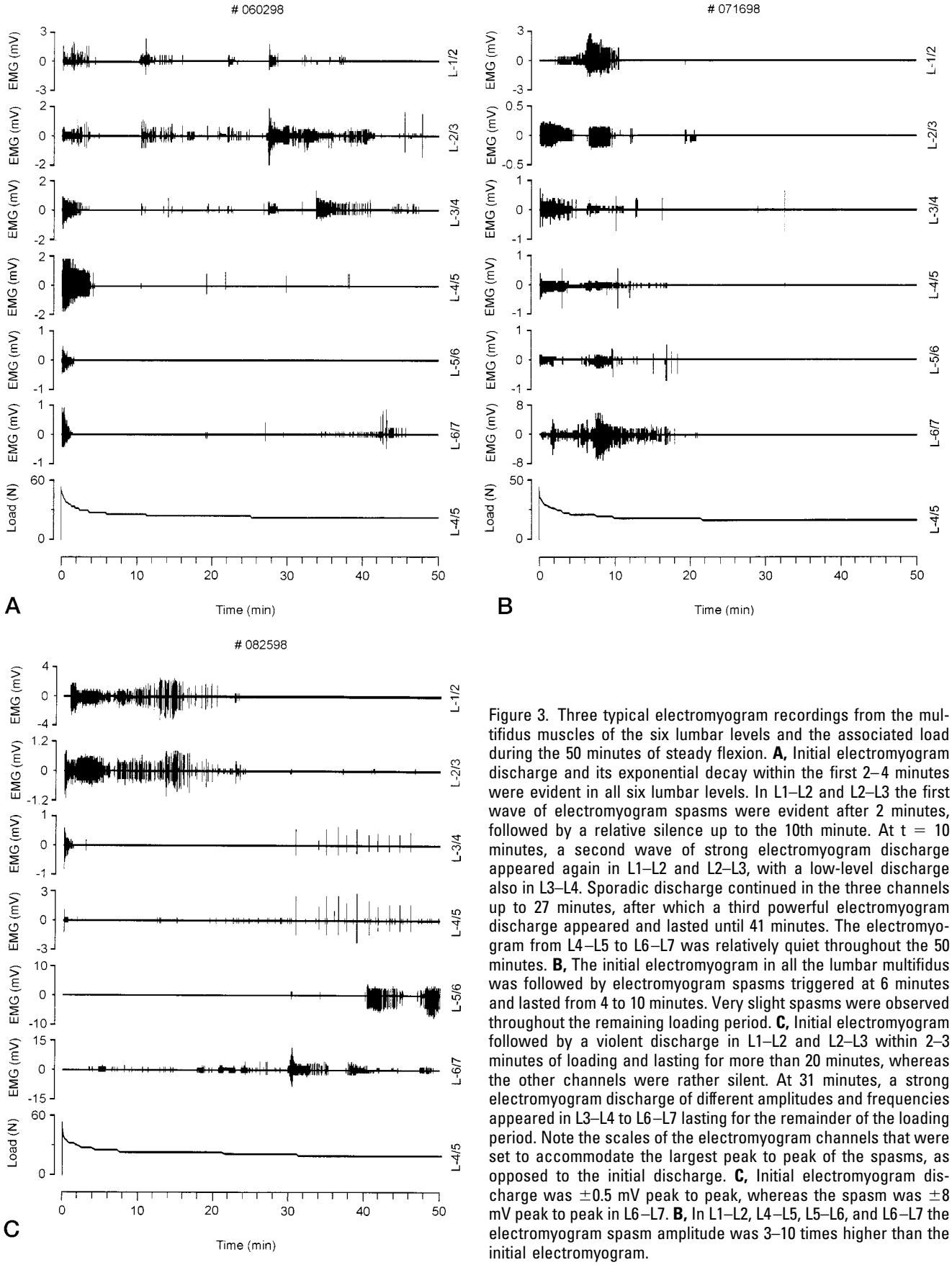

Figure 3. Three typical electromyogram recordings from the multifidus muscles of the six lumbar levels and the associated load during the 50 minutes of steady flexion. **A,** Initial electromyogram discharge and its exponential decay within the first 2–4 minutes were evident in all six lumbar levels. In L1–L2 and L2–L3 the first wave of electromyogram spasms were evident after 2 minutes, followed by a relative silence up to the 10th minute. At t = 10 minutes, a second wave of strong electromyogram discharge appeared again in L1–L2 and L2–L3, with a low-level discharge also in L3–L4. Sporadic discharge continued in the three channels up to 27 minutes, after which a third powerful electromyogram discharge appeared and lasted until 41 minutes. The electromyogram from L4–L5 to L6–L7 was relatively quiet throughout the 50 minutes. **B,** The initial electromyogram in all the lumbar multifidus was followed by electromyogram spasms triggered at 6 minutes and lasted from 4 to 10 minutes. Very slight spasms were observed throughout the remaining loading period. **C,** Initial electromyogram followed by a violent discharge in L1–L2 and L2–L3 within 2–3 minutes of loading and lasting for more than 20 minutes, whereas the other channels were rather silent. At 31 minutes, a strong electromyogram discharge of different amplitudes and frequencies appeared in L3–L4 to L6–L7 lasting for the remainder of the loading period. Note the scales of the electromyogram channels that were set to accommodate the largest peak to peak of the spasms, as opposed to the initial discharge. **C,** Initial electromyogram discharge was ±0.5 mV peak to peak, whereas the spasm was ±8 mV peak to peak in L6–L7. **B,** In L1–L2, L4–L5, L5–L6, and L6–L7 the electromyogram spasm amplitude was 3–10 times higher than the initial electromyogram.

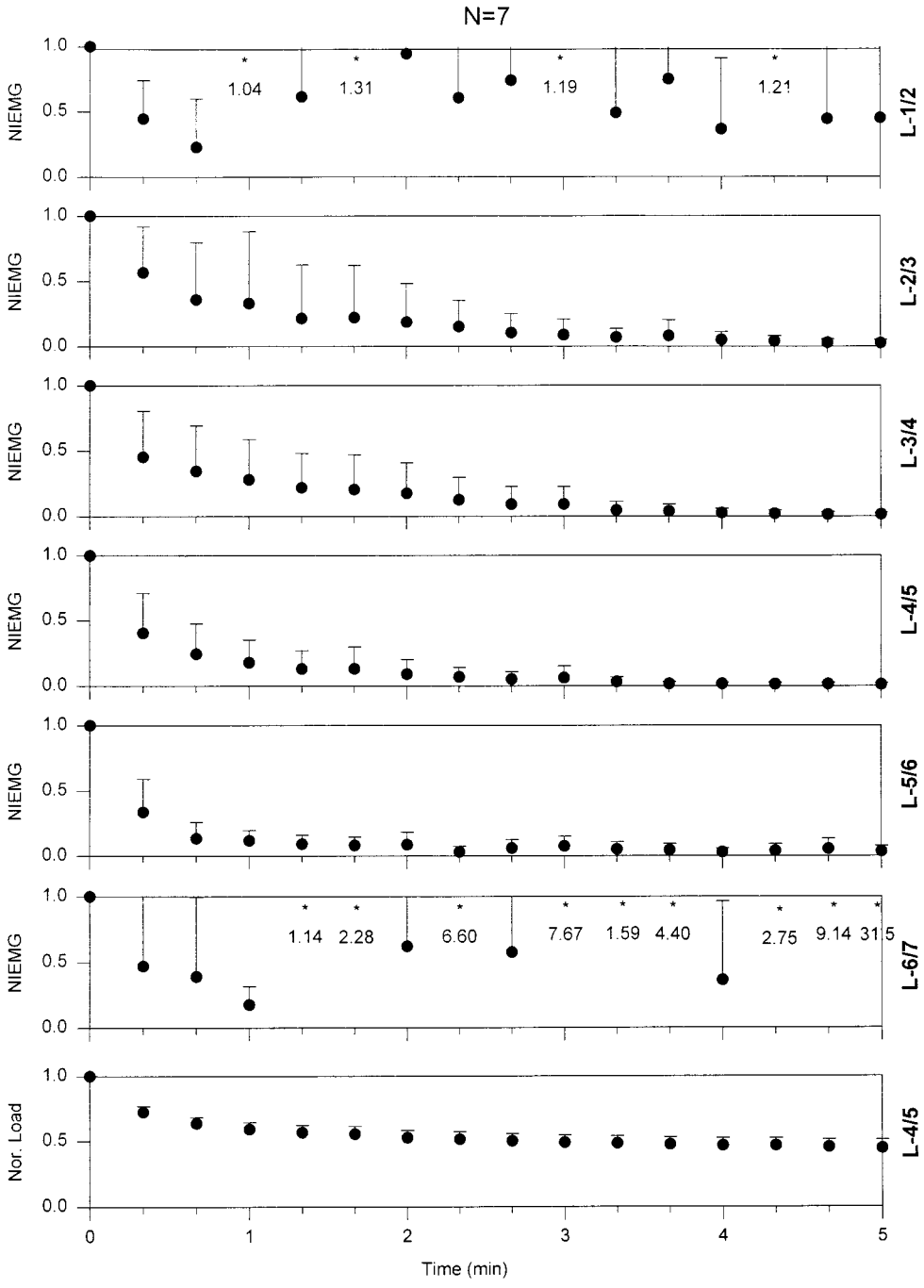

Figure 4. Mean ± SD of the normalized integrated electromyogram (NIEMG) recorded from each lumbar level of all seven preparations, along with the load measured at L4–L5, for the first 5 minutes of the 50-minute loading period. An exponential decrease of the NIEMG was present in all channels, diminishing to 5% of its initial value within 2–3 minutes. In L1–L2 and L6–L7, the exponential decrease in the NIEMG was interrupted by spasm activity within 1–2 minutes of loading, reflecting the excessive loading and possible early damage to the viscoelastic tissues of this level.

ter. At the end of the 50-minute period, the normalized load was 40% of its initial value.

Similarly, the mean NIEMG recorded from the multifidus of L2–L3 to L5–L6 decreased in an exponential manner, diminishing to 5% of its initial value within the third minute of loading. In the multifidus of L1–L2 and L6–L7, a similar exponential decrease was observed during the first minute of loading, after which it was interrupted by electromyogram spasms that had an early onset on these most rostral and caudal motion segments (further discussed in the next section).

The mean (±SD) length of the L4–L5 interval (Lo) subjected to a 0.5-N preload was 19 ± 0.89 mm before and 19 ± 1.1 mm after the 50-minute loading session. The mean vertical displacement of the load cell to offset the effect of the laxity in the supraspinous ligament by a load of 0.5 N after the 50-minute loading period was 3.8 ± 0.57 mm, which is calculated (see Appendix B) to result in a 7.85% ± 2.3% residual axial strain in the supraspinous ligament, a direct manifestation of the 50 minutes of displacement.

■ Discussion

The major findings of this investigation were that prolonged static flexion of the lumbar spine resulted in initial reflexive activity of the multifidus which diminished

within the first 3 minutes as tension-relaxation and laxity began in the viscoelastic structures, after which random electromyogram activity (*e.g.*, spasms) occurred in the same muscles. The lumbar levels in which multifidus spasms were observed, as well as the amplitude and time of its appearance varied widely and did not establish a general pattern. Spasms were visually observed to spread to other muscles (spinalis and longissimus) as well, indicating that neurologic connection to mechanoreceptors in the viscoelastic structures is not limited to the multifidus.

Spasms and elevated activity of the lumbar muscles are commonly observed and electromyographically confirmed in patients with low back pain.[4,10,12,17,24,26] Reflexively induced spasms of the paraspinal and gluteal muscles were also shown to be directly related to crushing of the supraspinous ligament in the cat by Pedersen et al.[22] Pedersen's finding confirms the correlation between damage to viscoelastic tissues and spasms, whereas spasms are associated in humans with low back pain. It can therefore be assumed that injury to one or more of the viscoelastic structures is associated with pain and spasms in some of the musculature.

In the experimental paradigm used in the current study, the spine was displaced into moderate anterior flexion for a relatively long period. The displacement was within the physiologic range and was not intended to inflict deliberate damage on any one structure. It is conceivable that the strain in the viscoelastic tissues, however, accumulated over time to a magnitude that was substantial enough to cause damage[1] and initiate reflexive spasms and probably pain. Pain could not be assessed in the anesthetized animal.

Idiopathic low back pain has been shown to arise from structural damage in several organs.[18] This includes excessive strain or deformation of ligaments and discs, deformation of the facet joint and its capsule, and compression of nerve roots as some of the most common problems associated with occupational activities consisting of repeated motion or prolonged static posture of the lumbar spine. In fact, the deformation and subacute damage of any of the described anatomic structures does not have to be substantial enough to be clearly observed with routine radiographic examination (such as rupture of ligaments or disc prolapse). Many patients with low back pain in this diagnostic category have actual recovery with rest, with the pain and spasms lessening as the respective damaged structure heals with time and no exposure to additional strain. It is possible that the source of the pain is from sensory receptors, which are known to exist in the various ligaments, discs, and joint capsules. Such receptors consist of two distinct groups: mechanoreceptors, which monitor load, pressure, position, vibrations, and motion, and nociceptors, which indicate tissue damage.

The initial reflexive response of the muscles to the deformation of the spine was most likely initiated by the large mechanoreceptors (*e.g.*, Golgi, Pacinian, and Ruffini) which have low thresholds to strain in the tissue. The spasms recorded from the muscles, however, were probably elicited by the small-fibered nociceptors, which have a high threshold to mechanical deformation. The strain induced in the viscoelastic tissues must have accumulated over time, causing damage that triggered the nociceptors' activity. Indeed, the residual strain measured confirms that at least the supraspinous ligament had undergone significant strain that remained well past the flexion period. Follow-up work in our laboratory shows that the residual strain of 7.85% ± 2.3% did not fully recover, even after 8 hours of rest. Modeling of the recovery pattern also indicates that more than 24 hours of rest did not allow full recovery of the strain that developed in the ligament because of the 50 minutes of static flexion.

Orthopedically, long-lasting overstretching of ligaments is known as a sprain and is always accompanied by pain and very often by spasms. The common treatment of such disorders is prevention of exposure of the ligament to excessive stretching and rest. It seems that such disorders (*e.g.*, ligament sprain and associated pain and spasms) can occur in the spine as well, and that prolonged rest may constitute one effective treatment.

An important point is that once the spasms were initiated, they were not limited to the multifidus muscles. Distinct twitches were observed in the spinalis and longissimus muscles. Neurologically, the reflex arc from the viscoelastic tissues of the spine is probably much more complex than was first thought.[28,29] At physiologic ranges of motion it seems to recruit the limited muscle activity that may be necessary to create sufficient stiffness in the respective motion segment so that stability is maintained. Once the accumulated strain developed in the tissues is severe enough to cause damage, a more global reflexive activation of several muscles is evident. Recently, Indahl et al[14] demonstrated that such reflex muscular activation also recruits the longissimus muscle. It is clear that much more work is needed to elucidate the full scope of this interesting reflexive mechanism.

That reflexive activation of the musculature was limited to the multifidus muscles when the viscoelastic structures were strained within their physiologic limits (before accruing any damage due to the prolonged period of deformation) reinforces the issue of the uniqueness of their function. The role of the multifidus as the posterior stabilizer of the spine was proposed before by several investigators in humans and by using animal modeling techniques.[2,3,7,11,15,23,30] The data presented in the current study and in the authors' previous work also provide neurophysiologic (in addition to the clinical and biomechanical) evidence that the unique role of the multifidus is to maintain spinal stability.

The observation that spasms were also detected in the longissimus and spinalis once damage was inflicted in the viscoelastic structures, expands the role of stability to more superficial muscles. Indeed, lumbar models predict that nearly all paraspinal muscles can contribute to stability, and the multifidus may have the strongest impact

in this role.[3,7] Recruitment of the spinalis and longissimus, however, is probably kept as a second line of defense, once some damage is present.

It should be pointed out that under the experimental conditions used in the current study, the lumbar spine was isolated by external fixation of the L1 and L7 vertebrae. Therefore the displacement applied to the center of the lumbar spine through the L4–L5 supraspinous ligament simulated moderate anterior flexion during which each motion segment shifted position naturally to accommodate the overall resultant flexion angle. The extreme motion segments (L1 and L7), however, were forced to remain near their resting position by the external fixators, creating the opposite effect (*i.e.*, the posterior ligaments of middle lumbar vertebrae were strained, and the anterior portion of each disc was compressed (see Figures 1 and 2). In the most rostral and caudal vertebrae, however, the anterior ligaments were strained, and the posterior portion of the discs were compressed (such as in a backward flexion). This may have caused a different pattern of strain in the most rostral and caudal tissues. Therefore, the early and frequent appearance of spasms in the multifidus of L1–L2 and L6–L7 could be explained by the different strain pattern and possible early strain damage in the tissues of those motion segments, as is evident in Figure 4. The figure shows that the initial decay of reflexive muscle activity was interrupted by spasms within 1 minute of loading.

Figure 4 points out that the load-relaxation associated with the prolonged flexion of the lumbar spine decreased to less than 50% of its initial value within the first 3 minutes and continued to diminish slowly with time. In a previous study,[27] the lumbar spine was subjected to cyclic flexion at 0.25 Hz and similar displacement amplitudes (*e.g.*, 12–15 mm from resting position). The load associated with the cyclic flexion also diminished exponentially with time, but at a slower pace. A decrease to 61% of its original load was observed after 3 minutes of cyclic flexion, and reduction to 50% occurred in 15 minutes. Electromyogram spasms were never observed in the cyclic flexion experiments. Does damage to the viscoelastic tissues and the consequent muscular spasms depend on the rate at which strain is applied? This issue needs in-depth investigation, because it may have significant implications in the design of optimal work and rest periods in occupational activities.

The fast decay in the reflexive muscular activity of the multifidus muscles indicates that maintaining a specific nonneutral stationary posture for a prolonged period depletes the spine of the stiffening and associated intervertebral stability provided by the musculature. With the loss of such muscular forces, the spine may be subjected to increased exposure to destabilizing injury. Such increased exposure to instability is compounded by the laxity of the viscoelastic structures. In fact, muscular spasms, probably associated with pain, can result even without injury.

That the force sustained by the viscoelastic structures through the supraspinous ligament decreased to 50% of its initial value within 3 minutes, and that a 95% reduction in the reflexive muscular activity occurred in the same period was surprising. It was expected that the force and the electromyogram would decrease much slower, arriving at a nonfunctional state in 15–20 minutes, which is the duration most of the laboratory staff found to produce discomfort in a brief study in which deep anterior flexion was performed. However, it must be remembered that the data presented in this study were from the cat, which is a quadruped. In four-legged models, the posterior viscoelastic tissues are not as developed as in the biped human and offer much less resistance to stretch and probably exhibit a faster rate of strain. In human, the cross-section of the posterior ligaments is much larger and would probably exhibit less laxity with time and therefore would sustain reflexive muscular activity for longer periods before diminishing to a nonfunctional level, undergoing subacute damage and initiating spastic activity in the musculature. The general pattern of responses, however, is expected to be similar in human.

■ Conclusions

Based on the results of this investigation, the following conclusions could be made: Prolonged static flexion of the lumbar spine elicits reflexive contraction of the multifidus muscles as they attempt to maintain intervertebral stability. Tension-relaxation and laxity in the viscoelastic tissues, set by prolonged flexion, desensitizes the mechanoreceptors and causes an exponential decrease in the contraction level of the multifidus muscles. In the cat, a 95% decrease in electromyogram activity was evident within 3 minutes of steady deformation and coincided with 50% loss of the load sustained by the viscoelastic structures. Electromyogram spasms are associated with prolonged static flexion of the lumbar spine. The electromyogram spasms appeared at any given time during flexion, past the first 1–2 minutes, and at any of the lumbar levels. Several waves of spasms in the same muscles can occur and sometimes long after the load has been removed. The muscular spasms elicited by the prolonged static flexion of the lumbar spine are not limited to the multifidus; they were observed in the spinalis and longissimus muscles as well. It is suggested that strain of viscoelastic tissues accumulates over time to cause subacute damage that causes nociceptors to reflexively elicit spasms in the musculature, probably associated with the sensation of pain.

In summary, direct experimental evidence is provided linking prolonged static flexion of the spine with muscular spasms that are characteristic of low back pain and various associated disorders.[8,15,19,25] The results of this study also confirm that prolonged flexion of the spine results in diminished muscular activity, which is associated with decreased stability.

The results of this study suggest a new paradigm for occupational low back pain in individuals subjected to

prolonged static nonneutral spinal postures within physiologic range and associated with negative radiologic findings (such as prolapsed disc and ruptured ligaments). In such cases, severe strain of the viscoelastic structures develops over time, inflicting subacute damage. Nociceptors in the damaged viscoelastic structures reflexively initiate spasms in the musculature while eliciting pain sensation. The pain and spasms may last as long as the damage persists and slowly disappear as rest allows the damaged structures to recover.

Previously, it was thought that prolonged assumption of a given posture is associated with muscle overuse, fatigue, and ischemic conditions, which in turn initiate the sensation of pain and spasms. The findings in study showed, however, that the same symptoms could occur when the muscles are not voluntarily contracting (anesthetized cats), excluding muscle overuse and fatigue, thereby lending support to the new hypothesis of low back pain of the cited cause.

■ Key Points

- Prolonged static flexion of the lumbar spine results in tension-relaxation and long-lasting residual strain of its viscoelastic structures.
- Reflexive electromyographic activity from the multifidus muscles initially decreases, and then exhibits spasms across all the lumbar levels throughout the flexion period.
- It is understood that in viscoelastic structures, subacute damage (*e.g.*, sprain), which develops during prolonged static flexion, excites nociceptors that give rise to spasms.
- Prolonged static flexion exposes the spine to instability and could result in low back disorders due to subacute damage to ligaments or discs.

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■ Appendix A

The physiologic strain in the L1–L2 to L6–L7 supraspinous ligaments was determined as follows: Short hypodermic needles were inserted into the dorsal process of each lumbar vertebrae, and the length from needle to needle recorded with calipers in two conditions. In the first condition the preparation was laid prone during the measurement, and that was considered the neutral condition. In the second condition the preparation was laid on its side and flexed passively to simulate a cat in a common sleeping position with its head near its hind limbs, and a second measurement was taken to constitute a near maximal strain condition. The physiologic strains were calculated as the percentage of the elongation from the neutral condition and are shown in Table A1.

The range of physiologic strains varied from 4.3% to 42.5% in the different lumbar levels and presented no new or different information from that presented by Panjabi et al[20] in 1982 for human lumbar ligaments.

Measurements of the L4–L5 dorsal process to process length, as well as the vertical displacement of the S-shaped hook from the L4–L5 baseline allowed calcula-

Table A1. Physiologic Strains in the Lumbar Supraspinal Ligaments of the Feline

N = 10

Level	Resting (mm)	Max Flex (mm)	Strain (%)	Range (%)
L ½	15.0 ± 1.4	16.9 ± 1.7	12.9 ± 9.8	4.3 - 22.3
L 2/3	16.0 ± 1.5	19.0 ± 3.0	19.1 ± 9.8	4.5 - 27.2
L 3/4	18.3 ± 1.5	22.2 ± 2.8	21.3 ± 7.6	7.2 - 24.4
L 4/5	19.0 ± 0.5	23.4 ± 2.7	23.6 ± 12.2	14.7 - 42.5
L 5/6	20.0 ± 2.5	22.9 ± 3.8	14.4 ± 6.0	11.6 - 23.9
L 6/7	20.8 ± 2.6	23.4 ± 2.9	12.7 ± 1.7	10.3 - 14.2

tions of the L4–L5 supraspinous ligament axial length (see Appendix B) which was kept below 15% to ensure physiologic strain.

■ Appendix B

Estimation of the residual strain in the L4–L5 supraspinous ligament at the end of the 50-minute lumbar flexion (Figure B1), where Lo is length of the supraspinous ligament at 0.5 N before the 50 minutes of loading, Lf is length of the supraspinous ligament at 0.5 N after 50 minutes of loading, and Vd is vertical displacement of the load cell needed to elicit a 0.5-N load immediately after 50 minutes of loading.

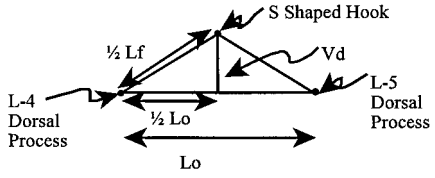

Figure B1.

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

and

$$Residual\ Strain = \frac{Lf - Lo}{Lo} * 100\% \qquad (2)$$

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