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Neuromuscular neutral zones sensitivity to lumbar displacement rate

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Abstract

Objectives. To determine the displacement and tension thresholds (developed during anterior lumbar flexion) which trigger reflexive muscular activity in the multifidus muscles; their variability with the velocity of flexion; and the pattern of threshold variability across the lumbar spine.

Design. An in-vivo study of the feline during passive lumbar flexion applied via the L-4/5 supraspinous ligament.

Method. EMG from six pairs of intramuscular electrodes inserted in the L-1/2 to L-6/7 multifidus muscles was recorded while the lumbar spine was passively flexed to 75% of the physiological strain of the supraspinous ligament at rates of 17–100%/s. Three-dimensional models of tension threshold, flexion rate and lumbar levels were developed from the experimental data.

Results. Displacement and tension thresholds were the lowest at the fastest flexion rate and gradually increased as flexion rates decreased. Electromyographic activity was detected at low thresholds at the center of the flexion and at gradually increasing thresholds at higher and lower lumbar segments.

Conclusion. Multifidus reflexive muscular activity, which stabilize the spine, is triggered at a displacement and tension thresholds of 5–15% of the physiological range. Earlier activation of muscular activity occurs as the velocity of flexion increases. Earlier activation also occurs near the center of flexion.

Relevance

Sensory-motor neurological feedback maintains spine stability and is responsive to the velocity of lumbar motion. A neuromuscular silence exists in small lumbar movements in which spine stability is not protected by the musculature. Spine models constructed to predict risk factors could benefit from incorporating this new information.

Keywords: Spine; Muscle; Low back pain; Reflex

1. Introduction

It is established that the lumbar ligaments have a relatively limited direct role in regulating intervertebral stability [1–4], whereas the musculature functions as the major structure that maintains such stability in many conditions via the co-activation principle [3,5–11].

The sensory-motor relationships between ligaments and musculature in regulating the stability of limb and spine joints has been a gradually emerging concept for nearly a century. Payr [12] in 1900, suspected that ligaments may be endowed with various afferent elements that could monitor elongations and tensions and trigger reflexive muscular activity to support the joint. Payr's ''kinetic chain'' Hypothesis was elaborated upon by several researchers in the first half of the century [13–15] and more recently in our laboratory [16–19] and elsewhere [20,21]. In general, various afferents were found in the ligaments of the knee, elbow, ankle, shoulder and spine. In the spine, afferents are also present in the disc and facet capsule [22–25]. Electrical stimulation or direct application of tension/elongation to the various lumbar viscoelastic structures (ligaments, discs and capsules in the spine) were repeatedly shown to trigger activity in muscles that biomechanically tend to stabilize the given joint [26–30].

One issue that remains unclear is whether such reflex muscular activity is ongoing at all times or does it re-

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quire minimal necessary conditions to trigger (e.g., are there specific tension or elongation thresholds at which the afferents trigger muscular responses?) Solomonow et al. [16] observed that relatively high tension had to be developed in the isolated anterior cruciate ligament before the hamstrings contracted reflexively. In this experiment, the bones were externally fixed, and only the ligament was loaded. Conversely, when the same ligament was loaded while allowing afferents in other nearby structures (collateral ligaments, capsule, etc.) to be active, as in normal activities, the tension thresholds at which muscular activity was triggered were significantly lower [20]. This indicates that substantial amount of sensory integration from several sources may be taking place to provide joint stability across most of its range of motion.

Similarly, our recent work [29] shows that mechanical stimulation of the isolated supraspinous ligament of a single motion segment triggers electromyographic (EMG) activity in the multifidus at significantly higher tension levels as compared to mechanical stimulation of the same ligament segment together with afferent inputs from other nearby ligaments and discs. Although muscular activity was triggered at much lower tension/elongation levels of the ligament, a certain threshold was still observed. Tension or elongation in the ligament below that threshold did not trigger muscular activity.

Panjabi [31] observed that intervertebral motion near the normal resting position offers minimal resistance to displacement and defined it as a "neutral zone". Displacement beyond that range met with greater resistance and could prevent mechanical instability and injury. He went on to show that such "mechanical neutral zones" (MNZ) could be decreased with exerted muscle forces [32].

The information available so far indicates that substantial interaction of afferents in different viscoelastic structures, and possibly muscles [33,34], exists on an ongoing basis for proprioceptive (subconscious feedback control of movement such as joint stability) and kinesthetic (cognitive perception of movement) functions. The issue of the existence of such sensory-motor feedback in small perturbations around a resting position, or the threshold at which such sensory-motor feedback becomes active still needs much clarification. From the work of Solomonow [16,29], Sjolander [20] and Panjabi [31], it also emerges that mechanical neutral zones and "neuromuscular neutral zones" (NNZ) (the lumbar displacement or tension threshold below which muscles remain reflexively inactive) may co-exist, and that spatial or temporal relationships between them are likely, but are unknown.

Finally, since the afferents are within the spinal viscoelastic structures, and since the tension or elongation developed by such viscoelastic structures is dependent on the rate at which the stimulus is applied, we hypothesize that the NNZ may vary with the rate of viscoelastic tension development or elongation. The relationships between the rate of viscoelastic tension development or elongation and the initiation of reflexive muscular activity is also unknown.

The objective of this investigation is to provide new insights to some of the issues raised by the above hypothesis. In specific, we attempt to answer the following questions: What are the NNZs in the lumbar spine with combined inputs from afferents in various lumbar viscoelastic structures? Are the NNZs sensitive to the rate at which tension/elongation is developed in ligaments, discs, etc.? And what is the activity pattern of the neuromuscular neutral zones along different lumbar levels?

2. Methods

2.1. Preparation

Six adult cats weighing 4.7 (SD 0.44) kg anesthetized with a single dose of chloralose (60 mg/kg), were used 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 to expose the intact dorsolumbar fascia. The preparation was placed in a rigid stainless steel frame that allowed the isolation of various lumbar levels via external fixation. The external fixation did not prevent micro or macro motion in the lumbar spine, and was intended to avoid interaction with thoracic and sacral structures.

2.2. Instrumentation

Six pairs of fine stainless steel wire EMG electrodes, insulated except for a 1 mm exposed tip, were inserted 3–4 mm apart into the multifidus muscles of the L-1/2 to L-6/7 (cat has seven lumbar vertebrae) on the right side 6–8 mm from the midline. Each electrode pair constituted the input to a differential EMG amplifier with a 110 db common mode rejection ratio, a gain of up to 200,000, and a band pass filter in the range of 6–500 Hz. A ground electrode was inserted elsewhere in the preparation. The EMG response from each of the six channels was monitored on the oscilloscope, recorded and stored in a computer with a sampling rate of 1000 Hz.

An S-shaped stainless steel hook was inserted around the middle of the L-4/5 supraspinous ligament and connected to the cross-head of the Bionix 858 Material Testing System (MTS, St. Paul, MN, USA), which was instrumented with a computer controlled displacement system. The output of a force transducer placed in the displacement armature was sampled into the computer along with its displacement. The overall set-up is shown schematically in Fig. 1.

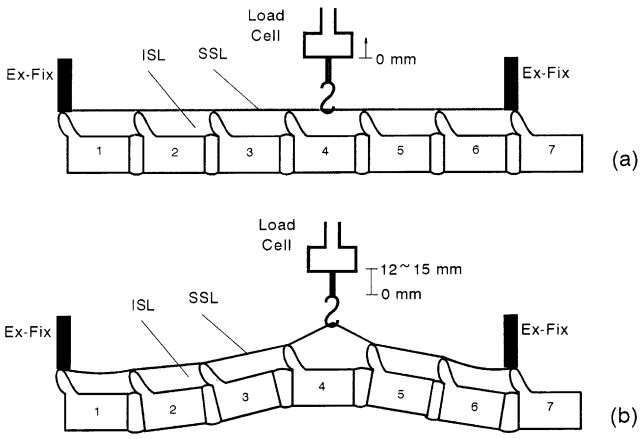

Fig. 1. A schematic of the experimental set-up depicting the lumbar spine and the displacement arrangement via the L-4/5 ligament with the external fixators applied to the L-1 and L-7 dorsal processes at rest (a), and during the displacement (b). Note that ligaments, discs and capsular tissues of several vertebrae were exposed to changes as the lumbar spine flexed.

In summary, EMG from six lumbar levels, displacement and tension data were recorded and stored in the computer for later analysis.

2.3. Protocol

In order to assert that several ligaments, discs and capsules were deformed by the flexing lumbar spine subjected to the passive displacement, one radiograph was made in the resting position and a second one during full displacement.

One external fixator was applied to the L-1 dorsal process, and a second one to the L-7 dorsal process. The stainless steel hook was attached to the cross-head of the Bionix 858 System which was set to deliver ramp displacements of 12–15 mm peak (according to the animal's size) with a pre-tension of 0.5 N. Vertical displacement of 12–15 mm were shown in our previous work [35] to result in ~15% axial strain of the supraspinous ligament which is about 75% of its maximal physiological strain (see Appendices A and B). A pre-tension of 0.5 N was selected as it standardizes the initial conditions across all preparations, while having very little effect, if any, on the supraspinous ligament's initial elongation/tension.

Initially, five sinusoidal cycles at 0.25 Hz and 12–15 mm peak displacement were applied in order to allow the preparation to settle into a steady-state condition, to assess and prevent any motion artifacts or instabilities at the fixation points, and to assess the integrity of noise free EMG recordings in all six channels. Following the five cycles, a 20-min rest period was observed to allow recovery of any tension–relaxation or creep that may have developed in the viscoelastic structures [36,37].

Ramp inputs consisted of linear increase in displacement from a zero position (at which pre-tension of 0.5 N was recorded) to full displacement (12–15 mm) over the following durations; 1, 2, 4, and 6 s. Such inputs corresponds to displacement rates of 100%/s, 50%/s, 25%/s and 16.7%/s, respectively (where 100%/s represents, for example, 12 mm/s, when the maximal displacement was 12 mm in a given preparation). Once the peak displacement was reached, it was maintained for 2 s and then returned to zero. In order to assess repeatability, each of the four ramps was repeated. A 20-min rest period was allowed between each recording to allow full recovery of any viscoelastic tension–relaxation and laxity that may have developed due to a previous trial [36,37].

The ramp displacement inputs were randomized in order to prevent any influence of input duration order on the results.

2.4. EMG thresholds analysis

The first 500 ms of each EMG record, which was prior to the initiation of movement by the actuator were used as a benchmark for baseline signal noise. The mean absolute value (MAV) of this baseline EMG was calculated by first full wave rectification and then smoothing with a low pass filter of 200 ms time constant. After this period, any point along the signal that exceeded 10 times the baseline MAV was denoted as the threshold of activity in that channel. The corresponding tension and displacement values were recorded as threshold values for that particular trial and channel. This automatic procedure was manually supervised, to insure that any unexpected signal artifacts were not detected as thresholds.

After thresholds were detected for all channels and all trials, they were pooled by EMG channel (corresponding to lumbar level) and ramp rate, and plotted as function of rate, as function of lumbar level, and as a three-dimensional function of rate and lumbar level. The two trials recorded for each rate were separately (not as a mean of two trials) included in the calculations described above such that any variability were reflected in the standard deviation of the mean. To assess the effect of rate and lumbar level on threshold loads and displacement, a two-way analysis of variance (ANOVA) was used.

2.5. Model development

Following the results of the two-way ANOVA, a two-dimensional partial regression model of the threshold loads and displacement was undertaken. Because of the potential effect of rate, lumbar level and interaction, the generalized model includes these effects. The resulting generalized model function takes the following form:

$$y(r,x) = a + f(r) + g(x) + h(rx),$$

where y is the threshold load or tension, r the ramp rate (1/s, range 0.17–1), x the lumbar level (1–6), a the constant threshold level independent of rate or level, $f(r)$ an undetermined function of rate – used if ANOVA shows an effect of rate, $g(x)$ an undetermined function of lumbar level – used if ANOVA shows an effect of lumbar level, and $h(rx)$ an undetermined function of rate times level – used if ANOVA shows an effect of interaction between level and rate.

3. Results

The radiographs taken during rest and full displacement of the lumbar spine are shown in Figs. 2(a) and (b), clearly demonstrating that several viscoelastic structures of several levels were stimulated mechanically by the flexion. A typical recording of displacement of the lumbar spine via the L-4/5 supraspinal ligament at a rate of 50%/s with the associated tension and EMG recorded from the L-1/2 to L-6/7 multifidus muscles is shown in Fig. 3. Arrows were superimposed on the experimental data to delineate the initiation of the EMG activity in each of the six channels. The vertical projection of each arrow to the corresponding displacement and tension curves yields the displacement threshold or tension threshold of the EMG activity.

In general, the EMG from the L-3/4 multifidus muscles appeared first, and the initiation of EMG in the higher and lower levels was delayed by 50–300 ms, and corresponded to higher displacement or tension threshold. In several instances, the EMG in the L-3/4 and L-4/5 appeared simultaneously or within an extremely short delay. The delay between the L-3/4 to the more remote levels, however, was consistently large among the different preparations.

Fig. 4(a) provides the graphical representation of the mean displacement threshold (pooled from the six

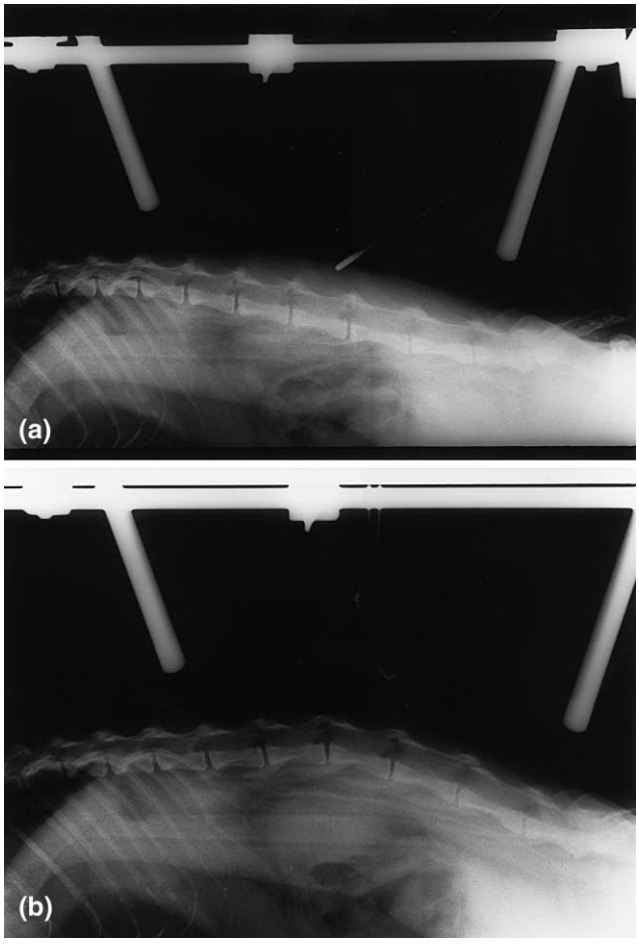

Fig. 2. Radiographs of the lumbar spine during rest (a) and during full flexion elicited by the displacement via the L-4/5 supraspinal ligament (b).

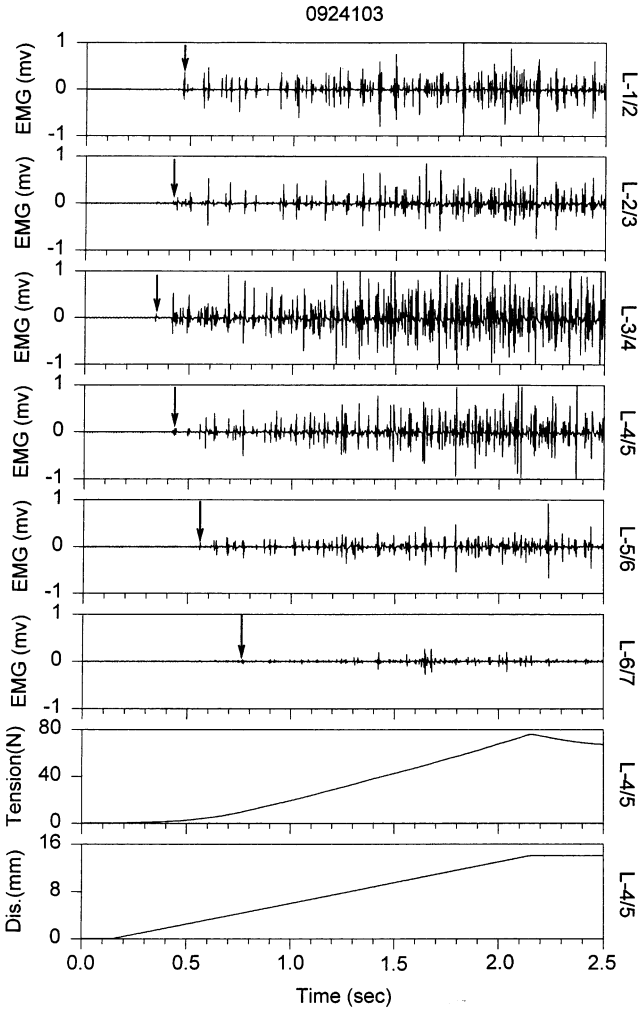

Fig. 3. Typical recordings of EMG from the multifidus muscles of six lumbar levels (top six traces), the tension and the applied displacement developed in the lumbar spine at a rate of 50%/s. Note the linear increase in displacement (bottom) dictated by the stimulus and the associated initial nonlinear rise in tension.

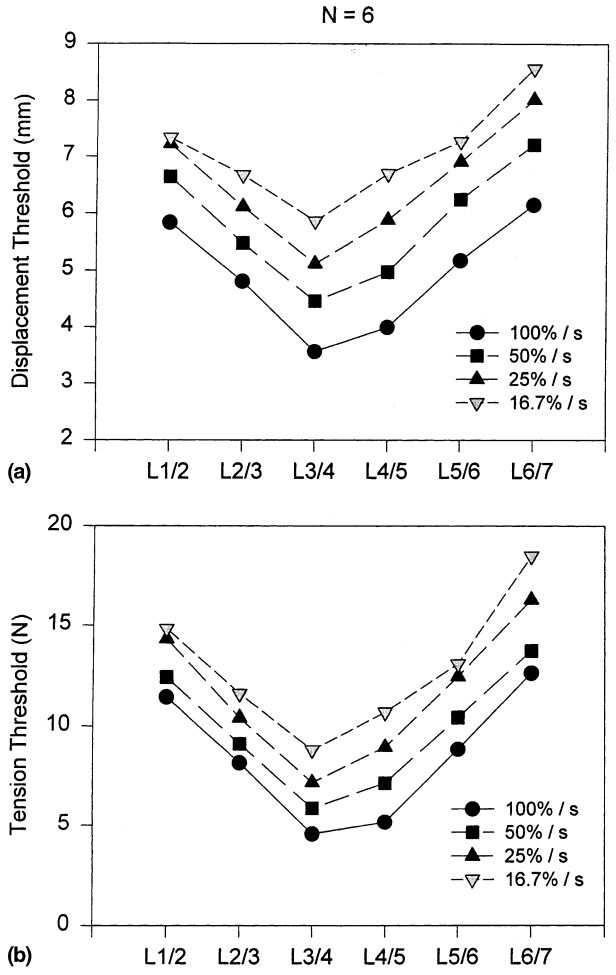

Fig. 4. (a) Presents the mean displacement threshold of each lumbar level for the four displacement rates applied to the spine. (b) Presents the mean tension threshold of each lumbar level for the four displacement rates. The mean values are based on the data from two trials at each rate from each preparation entered separately into the calculations.

preparations) vs the six lumbar EMG channels for the four displacement rates, whereas in Fig. 4(b) the mean tension threshold vs the six lumbar EMG channels is shown. Tables 1 and 2 provide the mean and standard deviation of the displacement and tension thresholds, respectively, for the four displacement rates employed in the study. The data are based on the two repeated trials taken at each rate.

The data shown in Fig. 4 indicate that both the displacement and tension thresholds were the lowest for the L-3/4 level, whereas the thresholds increased at higher and lower levels. This pattern was also consistent regardless of the displacement rate, except the fact that faster displacement rates had lower thresholds. Both the displacement and tension thresholds gradually increased as the displacement rates progressively decreased.

For a displacement rate of 100%/s, the lowest mean displacement threshold of 3.57 (SD 1.57) mm was recorded at the L-3/4 level and it gradually increased by 72.3% to 6.15 (SD 1.65) mm for the L-6/7 level. Similarly, a gradual increase of 63.6% to 5.84 (SD 3.06) mm at L-1/2 is present. The increases from the lowest displacement threshold at L-3/4 to that at L-6/7 for the displacement rates of 50%/s, 25%/s and 16.7%/s were 61.7%, 56.4% and 46.1%, respectively. The increase in displacement threshold from L-3/4 to L-1/2 for the displacement rates of 50%/s, 25%/s and 16.7%/s were 48.9%, 41% and 25.1%, respectively.

The lowest tension threshold of 4.56 (SD 2.77) N was at L-3/4 for a displacement rate of 100%/s and it increased to 12.61 (SD 5.73) N at L-6/7, a 176.5% change. Increases of 134.5%, 127.2% and 110.3% were calculated for displacement rates of 50%/s, 25%/s and 16.7%/s at the L-6/7 level, respectively. Increases of 150.6%, 112%, 104% and 68.7% were present at the L-1/2 level compared to that of L-3/4 for displacement rates of 100%/s, 50%/s, 25%/s and 16.7%/s, respectively.

Fig. 5 represents the data of Fig. 4, with the displacement and tension thresholds calculated as a percentage of the maximal displacement and tension, respectively, applied or recorded in the specific trial. Fig. 5 shows that displacements of about 25% of the maximal elicited the lowest EMG excitation threshold at L-3/4 at 100%/s rate, and that excitation thresholds of up to 60% of the maximal displacement applied were recorded at the most remote location, e.g., L-6/7, at 16.7%/s.

Similarly, tension thresholds of 7% of the maximal tension applied were required to elicit EMG at L-3/4 for 100%/s rate. The highest tension threshold of 41% of maximal was recorded at L-6/7 for the slowest rate of 16.7%/s.

Fig. 6 represents the same data of Fig. 4 in the form of the mean displacement thresholds (a) and the mean

Table 1
Mean (SD) of displacement threshold for the four displacement rates

Rate (%/s)	Displacement (mm)					
	L-1/2	L-2/3	L-3/4	L-4/5	L-5/6	L-6/7
16.7	7.33 (3.43)	6.67 (2.82)	5.86 (2.62)	6.70 (3.00)	7.27 (2.93)	8.56 (3.75)
25	7.22 (3.63)	6.12 (2.81)	5.12 (2.48)	5.89 (2.98)	6.91 (2.82)	8.01 (2.04)
50	6.64 (3.36)	5.48 (2.34)	4.46 (2.49)	4.98 (2.91)	6.24 (2.33)	7.21 (1.84)
100	5.84 (3.06)	4.81 (2.36)	3.57 (1.57)	4.00 (1.46)	5.18 (1.83)	6.15 (1.65)

Table 2
Mean and (SD) of tension threshold for the four displacement rates

Rate (%/s)	Tension (N)					
	L-1/2	L-2/3	L-3/4	L-4/5	L-5/6	L-6/7
16.7	14.83 (10.2)	11.60 (6.63)	8.79 (4.73)	10.68 (6.69)	13.07 (6.73)	18.49 (11.8)
25	14.32 (10.2)	10.40 (6.83)	7.16 (3.84)	8.94 (5.04)	12.44 (7.40)	16.27 (5.73)
50	12.40 (8.31)	9.10 (5.36)	5.85 (3.49)	7.14 (4.73)	10.41 (4.09)	13.72 (4.00)
100	11.43 (8.91)	8.15 (6.85)	4.56 (2.77)	5.16 (2.42)	8.83 (4.31)	12.61 (5.73)

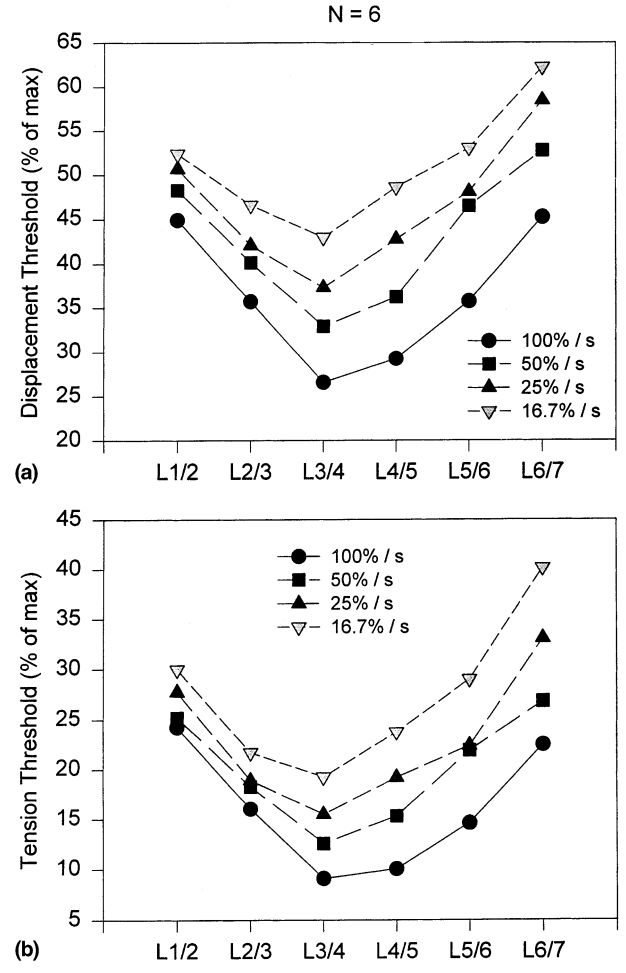

Fig. 5. The same data of Fig. 3 are plotted, except that the thresholds are plotted as the percentage of the corresponding maximal displacement and maximal tension obtained in each preparation.

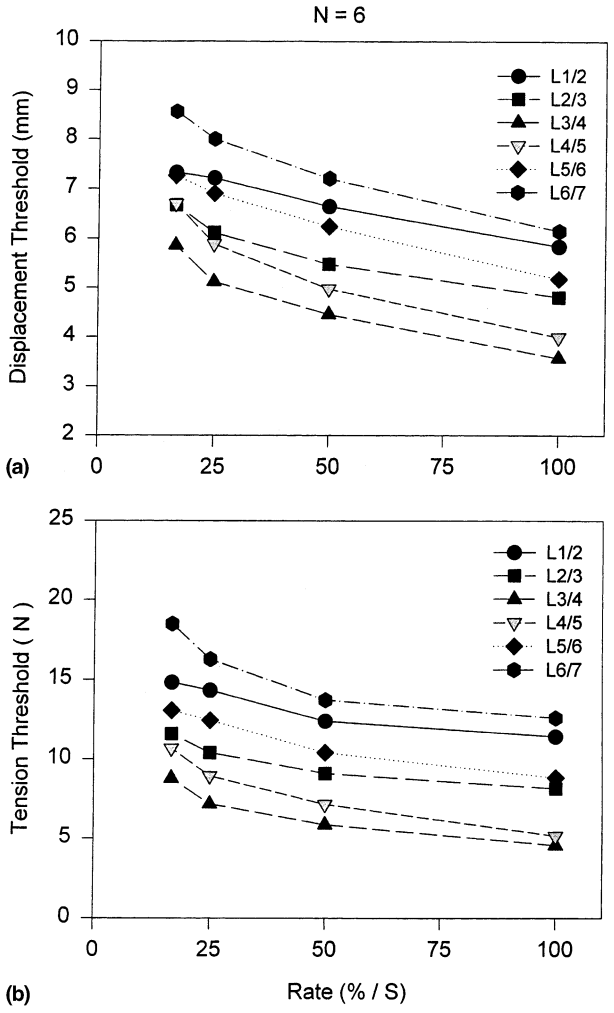

Fig. 6. The mean displacement threshold (a) is plotted against the displacement rate for each lumbar lever. In (b), the tension threshold vs displacement rate is plotted for each lumbar level.

tension thresholds (b) vs displacement rate for the six lumbar levels. The figure delineates the consistent pattern of decreasing displacement and tension thresholds as displacement rates increase from 16.7%/s to 100%/s.

4. Threshold analysis and model

The tension and displacement threshold values were analyzed by a two-way ANOVA, grouping by rate and lumbar level. For both tension and displacement, significant effect of rate ($P < 0.005$ and $P < 0.0001$, respectively) and of lumbar level ($P < 0.0001$ in both cases) were found. Similarly, no interactions were found between lumbar level and rate ($P > 0.999$ in both cases). This is interpreted as meaning that in modeling the relationship of threshold as a function of lumbar level and rate under these conditions, no cross-term describing

interaction is necessary. Hence, given the quasi-linear appearance of both tension and displacement with varying rate (Fig. 6) and the curves of thresholds with varying lumbar level (Fig. 4), we performed a partial regression of the mean threshold levels as linear function of rate plus second-order function of lumbar level. The resulting regression functions are of the following form:

$$y(r,x) = a + br + cx + dx^2,$$

where y is the threshold load or tension, r the ramp rate (1/s, range 0.167–1), x the lumbar level (L-1/2 to L-6/7), and a, b, c and d are the model regression coefficients.

The three-dimensional representation of the displacement threshold as a function of rate and lumbar level for the experimental data is shown in Fig. 7(a), whereas the graphical representation of the model derived above is given in Fig. 7(b). The vales of the coefficients a, b, c and d, as well as the correlation coefficient R^2 and SE for the experimental and model are given in Table 3.

Similarly, the three-dimensional model of the tension threshold as a function of rate and lumbar level based on the experimental data is shown in Fig. 8(a), and its derived model in Fig. 8 (b). The values of the coefficients a, b, c and d, the correlation coefficient R^2 and SE comparing the experimental data to its model are also given in Table 3.

5. Discussion

The major findings of this study indicate that the NNZ are relatively narrow and that there is a strong dependence of the NNZ on the rate at which the spine was flexed from its resting condition. Another finding is that the lumbar joints near the epicenter of the flexion are associated with a narrower NNZ compared to lumbar joints above and below, yet the neutral zones of all lumbar levels are sensitive to the rate of flexion. An inferential finding is that neuromuscular neutral zones are not fixed, but rather adaptive to the condition dictated by the specific type of the movement performed and its specific impact on a given lumbar level.

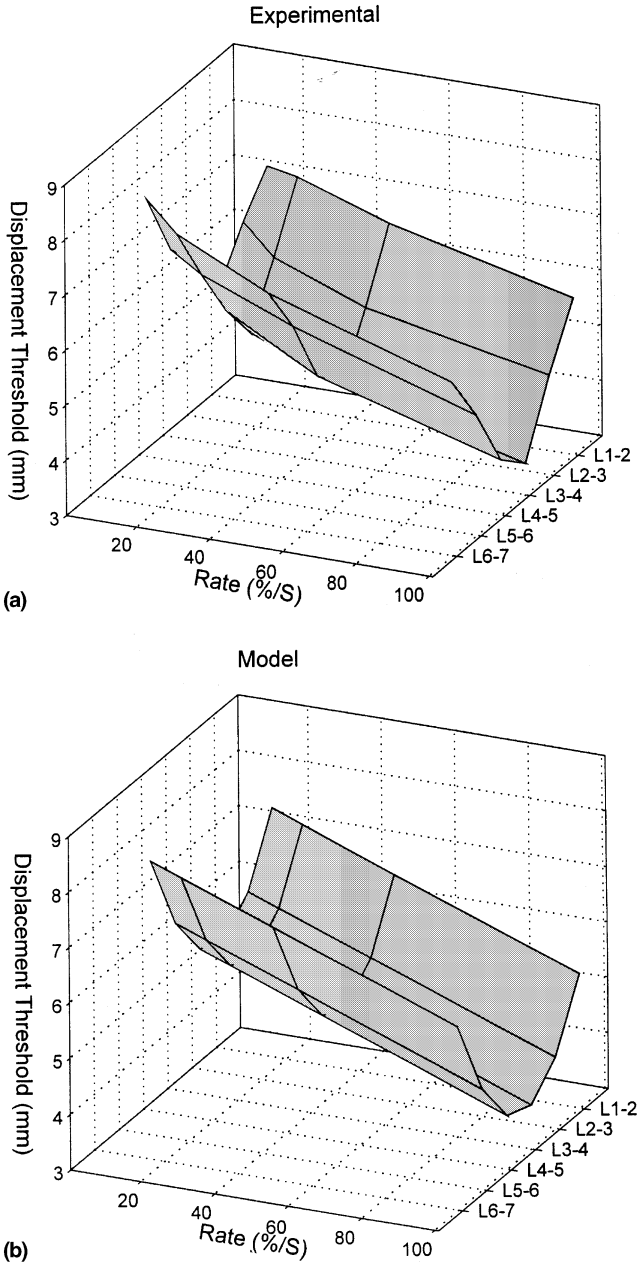

Fig. 7. A three-dimensional representation of the experimental displacement threshold as a function of displacement rate and lumbar level is given in (a), whereas in (b), the graphical representation of the displacement threshold model as a function of displacement rate and lumbar level is given.

Table 3
Regression coefficients and statistics for threshold model[a]

Parameter (y)	a	b	c	d	R^{2b}	SE[c]
Displacement	9.71	−2.50	−2.13	0.329	0.925	0.339
Tension	22.0	−4.94	−7.47	1.13	0.929	0.938

[a] $y(r,x) = a + br + cx + dx^2$, where y is the threshold load or tension, r the ramp rate (1/s, range 0.167–1), x the lumbar level (1–6) and a, b, c, and d are the model regression coefficients.
[b] The squared correlation coefficient.
[c] The model standard error.

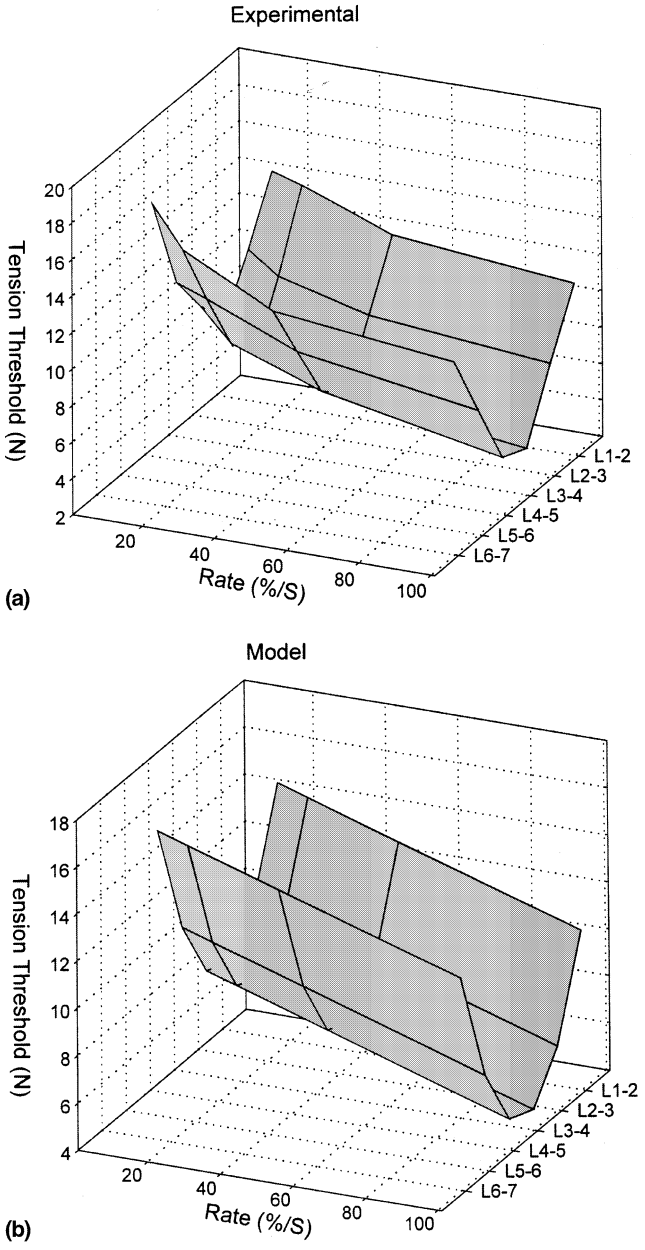

Fig. 8. The three-dimensional representation of the experimental tension threshold (a) and the model (b) developed for the experimental data.

It is important to recall that in the paradigm of this protocol passive lumbar flexion was elicited and that in turn deformed several ligaments, discs and capsules. The reflexive EMG, therefore, was triggered by multiple inputs from receptors in several structures (as opposed to inputs from receptors in a ligament of a single level). The NNZ, therefore, correspond to that of a system with multi inputs and multi outputs.

The fact that the neuromuscular neutral zones were sensitive to the rate at which the lumbar spine was flexed, is not surprising. There are three physiological mechanisms that can allow the nervous system to activate muscles while responding to the rate at which a movement is executed. It is well established that many mechanoreceptors are highly sensitive to the rate at which the tissue in which they are embedded is stimulated [38]. Encapsulated receptors, such as the Golgi and Pacinian, are known to be sensitive to the rate at which their substrate tissue is deformed or the load is applied. The Pacinian corpuscle, a fast adapting receptor, is especially sensitive to transient change of tension or elongation. The Golgi receptor is a slow adapting receptor, and can monitor changes as well as signal a static tension or elongation state for prolonged periods. Both Golgi and Pacinian afferents were found in the spinal ligaments, disc and capsule [24,25]. Furthermore, Ruffini afferents (also present in spinal viscoelastic structures) are also sensitive to the rates of tissue deformation. Since they are not encapsulated, their sensitivity to the rate of tissue deformation may be lower than that of the Pacinian afferent. Finally, the naked nerve endings found in the spinal structures are mostly nociceptors, with high threshold to stretch and force stimulus. Their nerve conduction velocity of action potentials is very low ($\leqslant$1 m/s) as compared to that of the afferents described above ($\geqslant$20 m/s). Overall, with high force or elongation threshold combined with a very low conduction velocity, the bare nerve endings are highly unlikely to be very sensitive to rates of stimulus application, and mostly reserved for nociception.

The second physiological mechanism which allows the nervous system to respond to rates of tissue loading is the architecture of the afferent distribution in the various tissues. In the human palmar wrist ligaments, for example, the radial collateral and the radiolunate ligaments have higher density of mechanoreceptors near their insertion into the respective bones. The distribution of mechanoreceptors is, however, nearly uniform throughout the radioscaphocapitate ligament [39]. Similarly, in the human elbow, the distribution of mechanoreceptors in the annular and transverse medial ligaments is uniform, whereas higher afferent density is found near the insertions of the radial, posterior and anterior medial ligaments [40]. In essence, afferents located in the compliant mid-substance of ligaments are much more sensitive to low stimulus thresholds and their rate of change with time as compared to afferents located near the much stiffer ligamentous insertion. Afferents in stiffer tissues will have higher threshold to stimulus magnitude as well as lower sensitivity to the rate of stimulus application. Such afferents are mostly suitable for signaling exceptionally high, or risky, loads that may require immediate assistance from the musculature in order to reestablish physiologically safe conditions (i.e., prevent damage to the tissues) [41]. Overall, ligaments with uniform distribution of afferents throughout their tissue are inherently highly sensitive to low loads and their rate of application.

The third mechanism which is known to respond to changes in the rate at which movement is executed as well as in formulating muscular response is the complex network of the central nervous system with the cerebellum being the primary component [42]. In this network, most peripheral afferent have inputs to the ascending spino-cellebellar tracts which terminates in the cerebellum [43]. In turn, the outputs of the cerebellum have powerful impact on the management of motor functions. Such inputs are applied by modulation of descending tracts in the brain stem, and by more indirect impact on spinal interneurons in modulation of spinal reflexes, including sensitivity to rate [38,42]. Therefore, the sensitivity of the neuromuscular neutral zones to changes of the rate of flexion of the lumbar spine is well founded in anatomical and physiological knowledge.

Co-activities in the antagonist musculature were shown to be triggered by afferents in ligaments in order to maintain stability [16]. Hagood et al. [44] also demonstrated that antagonist co-activity during isokinetic knee extension and flexion of normal healthy subjects was strongly influenced by the velocity of the movement. Higher knee velocities resulted in an initial decrease of co-activity level to allow limb acceleration and early increase of co-activity level before the end of the range of motion in order to provide dynamic braking. While the overall co-activity level is probably modulated by several other inputs, the work of Hagood [44] provides evidence of the complexity and adaptive properties of this sensory-motor feedback loop in much more complex conditions in behaving humans.

The sensitivity of the neuromuscular neutral zones to the rate of lumbar flexion has direct and straightforward biomechanical implications. Fast rates of flexion are associated with earlier and faster development of internal loads as well as larger forces associated with the higher acceleration [45]. Therefore, the reflexive muscular forces are triggered early and develop faster throughout the movement. Overall, the stability of the spine is maintained as may be required by the adaptive feedback of the reflexive activation of the musculature. The NNZ, therefore, increase and decrease with the rate of the associated movement. Since trunk velocity was identified as a risk factor [45], the ability or inability of the NNZ to adapt to velocity of motion in various conditions has to be subjected to further investigations. Of particular interest are the adoptive properties of the NNZ in cyclic activities as well as in sequential prolonged static flexions which are known to induce tension–relaxation or creep in the lumbar viscoelastic tissues, and diminish reflexive muscle activity [30,35].

In our previous work [35] it was determined that the axial physiological strain limits of the supraspinous ligaments are approximately 20–25% (See Appendix A). The 12–15 mm displacement applied to the L-4/5 segment in this study was chosen as it allowed nearly 75% of the physiological strain range of the L-4/5 supraspinous ligament to be explored. Accounting for the unexplored upper 25% of the physiological strain limits, one can conclude that the NNZ exist in the initial 5% of the tension and in the initial 15% of the displacement ranges. This information confirms the observation of Sjolander [20] and ours [29] that combined inputs from afferents in several viscoelastic structures associated with a joint result in triggering reflexive muscular activity at very low displacement or tension thresholds. One can conclude that reflexive muscular activity stabilize the spine once motion exceeds an extremely narrow neuromuscular neutral zone of about 5–15% of the full range.

Another issue that should be considered in perspective is the relatively lower tension threshold as compared to the corresponding displacement threshold as seen in Fig. 5. For the rate of 100%/s and at L-3/4, the tension threshold was below 10% of the maximal, whereas the displacement threshold was near 25% of the maximal. At face value, one can erroneously conclude that afferents in the lumbar viscoelastic structures are more sensitive to tension relative to displacement. In fact, close inspection of Fig. 3, shows that the displacement was increasing linearly throughout the test (bottom trace) whereas the tension exhibited an initial nonlinear rise (in the first 500 ms) followed by a nearly linear increase thereafter. This nonlinear force vs length curves of various viscoelastic structures (ligaments, tendons, etc.) is well established, and is the source of the misperception of preferential responsiveness to tension than to elongation. The time delay from the application of the stimulus to the appearance of EMG in the muscles was the same for both tension and displacement.

The lowest displacement and tension thresholds to the anterior displacement of the lumbar spine via the L-4/5 supraspinous ligament were recorded from the multifidus muscles of the L-3/4 level. Considering the fact that multifidus branches originating from any given lumbar vertebrae terminate on vertebrae 2–4 levels below, a considerable overlap exists. In our previous work [28,29], we found that there is a tendency of multifidus muscles to use the thoracic and pelvic levels as a reference structures from which stiffening forces are generated to stabilize the lumbar vertebrae. The thoracic spine is significantly stiffer (as compared to the lumbar spine) due to the ribs and the interconnecting musculature. It is only natural for the multifidus muscles originating from L-1 to L-4 to be more active, creating a stiffened lumbar segment extending to the L-4/5 level. Indeed, electrical and mechanical stimulation of the L-4/5 supraspinous ligament was found to induce more activity in rostral levels than in caudal [28, 29], and the same phenomena was seen in this study as well.

Since higher and lower levels were further away from the epicenter of the flexion, the tension and displacement developed in their viscoelastic structures were substan-

tially lower, hence the higher associated thresholds. Nevertheless, the complete lumbar spine was stiffened to some degree as response to the applied flexion at L-4/5 confirming the synergy of the function of the multifidus muscles and their primary role as lumbar stabilizers to anterior flexion [46].

Furthermore, while the higher and lower lumbar levels displayed higher displacement and tension thresholds relative to the L-3/4 level, the thresholds were always sensitive to the displacement rate. Lower thresholds were associated with faster displacement rate, indicating that the synergy of the multifidus of all levels maintains its integrity regardless of the velocity of the movement.

An important biomechanical issue is the fact that stiffening muscle forces are first developed near the epicenter of flexion, and as the flexion increases, multifidus muscles of the higher and lower levels stiffen their respective joints. The delay associated with triggering muscle activity at high and lower levels ranged from 50 to 300 ms. One can assume that as long as an intended function is performed uninterrupted, the stability afforded to the lumbar spine by the synergy of the lumbar multifidus is sufficient to prevent instability or injury. Sudden or fast interruptions of an intended movement, however, may not allow sufficient time for muscular reaction, and could result in instability and injury.

It is important to put the findings presented in this paper in the appropriate perspectives. The anterior flexion was applied to the center of the lumbar spine while it was passive, e.g., not during voluntary contraction. Such passive condition was an excellent background to determine the isolated pattern of response of reflexive muscular activity. The insight gained from the data provides valuable estimates of the general dynamics of the sensory-motor feedback of the lumbar spine. During voluntary daily activities of a human or animal, the dynamics of the sensory-motor feedback could be significantly modified as response to various external and internal factors. For example, a movement consisting of anterior flexion and rotation may result in a narrower NNZ in the multifidus of one side and a wider NNZ in the contralateral side. Based on the work of Hagood et al. [44] the position of a given vertebrae within its physiological range of motion and the prevailing joint velocity, combined with the external and internal loads, may further narrow or widen the NNZ as may be necessary in order to accomplish the intended motor task while minimizing the challenge to the stability of the spine. The general pattern of behavior described in this study, however, is still expected to persist, e.g., dependence of the NNZ on the velocity of movement and the distance from the epicenter of maximal flexion. These perspectives underscore the need for further exploration of the sensory-motor feedback dynamics in more complex conditions relevant to daily, sports and occupational activities.

Finally, the new information introduced in this paper may be useful in further development of spine models which predict risk factors associated with certain types of occupational activities and at certain external loads [3,9,10,45,47,48]. The incorporation of the NNZ, their pattern of activation across the lumbar levels and dependency on rate of flexion together with the known kinematic and dynamic lumbar behavior can expose and explain the sources of risk factors which were not clear and obvious before.

6. Conclusions

The following conclusions could be made based on the results of this study:

1. Neuromuscular Neutral Zones exist in the lumbar spine. It is estimated that such NNZ exist in the range of 5–15% of the maximal physiological displacement and tension generated by anterior flexion with an epicenter in L-4/5. Displacement and tension above 5–15% trigger activity in the multifidus muscles which affords stability in the lumbar spine. Lower values of displacement and tension are not associated with muscular activity.
2. The thresholds of the NNZ are dependent on the velocity of the movement. Flexions at a fast rate result in early onset of muscular activity whereas motion at slow velocity triggers muscular activity at a higher displacement or tension values. The NNZ are, therefore, adaptive with respect to the velocity of the movement.
3. The NNZ are relatively narrow at the epicenter of flexion, and gradually increase in lumbar levels above and below; yet all lumbar levels are responsive to velocity changes in a similar pattern (e.g., narrower NNZ for higher velocities).
4. A model describing the behavior of lumbar NNZ as a function of flexion velocity and lumbar level was developed and is anticipated to be useful for predicting risk factors when integrated in comprehensive dynamic spine models.

The information provided here allow a new insight into the synergy associated with the control of lumbar stability. The sensory-motor feedback loop originating from afferents in the various lumbar viscoelastic structures and executed by the musculature is evidently complex and adaptive to various conditions. The full extend of the dynamic behavior of such sensory-motor feedback warrants additional investigations in order to expose its full value in daily and occupational activities.

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Table 4
Physiologic strains (SD) in the lumbar supraspinal ligaments of the feline $N = 10$

Level	Resting (mm)	Max flex (mm)	Strain (%)	Range (%)
L 1/2	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

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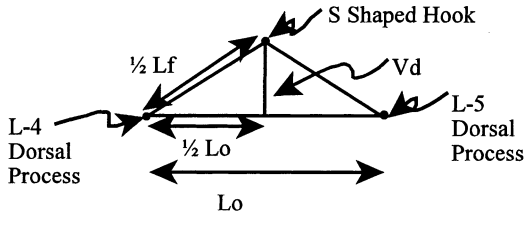
Appendix A

The physiologic strain in the L-1/2 to L-6/7 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 feline in a common sleeping condition with its head near its hind limbs, and a second measurement was taken to constitute near maximal strain condition. The physiologic strains were calculated as the percentage of the elongation from the neutral condition and are given in Table 4.

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

Appendix B

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



where Lo is the length of the supraspinous ligament at 0.5 N before 50 min of loading, Lf the length of the supraspinous ligament at 0.5 N after 50 min of loading, and Vd is the vertical displacement of the load cell required to elicit 0.5 N load immediately after 50 min of loading.

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

and

$$\text{Residual strain} = \frac{Lf - Lo}{Lo} * 100\%. \tag{B.2}$$

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