ABSTRACT: Neuromuscular control of lumbar stability following exposure to prolonged static work, under low and high loads, was assessed in the in vivo feline model. Six sessions of 10 min work at 20N with 10 min between rest was compared to a group subjected to the same protocol but carrying high loads of 60N. Displacement and tension developed in the spine at the instant the multifidus muscles applied stabilizing contractions, and their amplitudes were obtained from their electromyogram (EMG). Significant ($P < 0.001$) laxity developed in the various viscoelastic tissues of the lumbar spine that did not recover during and up to 7 h of rest postwork. Simultaneously, there was a significant ($P < 0.001$) decrease in muscular activity in the 3–4 h immediately postwork under low load but only during the first hour in the high load group. After that period the musculature compensated for the laxity of the viscoelastic tissues by a significant ($P < 0.001$) increase in activity in the high-load group and a nonsignificant increase in the low group. It was concluded that during 1–3 h immediately poststatic work a significant decrease in the stabilizing function of viscoelastic tissues together with a significant decrease in muscular activity is present, and they render the spine unstable and exposed to high risk of injury. Performance of prolonged static work under low loads, while not harmful during the work, cannot be designated as a "no-risk" condition, as it may result in injury postwork.

Muscle Nerve **39**: 71–82, 2009

NEUROMUSCULAR CONTROL OF LUMBAR INSTABILITY FOLLOWING STATIC WORK OF VARIOUS LOADS

BROOK LE, MSc,[1,2] **BRADLEY DAVIDSON, PhD,**[1] **DEBORAH SOLOMONOW, MSc,**[1]
BING HE ZHOU, EE,[1] **YUN LU, MD,**[1] **VIKAS PATEL, MD,**[1]
and MOSHE SOLOMONOW, PhD, MD(hon)[1,2]

[1] Musculoskeletal Disorders Research Laboratory, Bioengineering Division,
  Department of Orthopedic Surgery, Health Sciences Center, 12800 E. 19th Avenue,
  RC-1 N, Mail Stop 8343, PO Box 6511, Denver, Colorado 80045, USA
[2] Department of Electrical Engineering, University of Colorado,
  Denver, Colorado, USA

Occupational activities that require prolonged static lumbar flexion postures (flooring tiles and concrete work, farm work, auto mechanics, etc.) were shown by epidemiologic studies to be a risk factor for a cumulative low back disorder.[9,19,25,26] Handling loads of high magnitudes was shown to be especially conducive for low back disorders. Recent biomechanical and neurophysiological confirmation of the epidemiology identify substantial laxity/creep development in lumbar viscoelastic tissues concurrent with an acute neuromuscular disorder in in vivo models[2,15,25,30,31] and in humans.[3,6,7,14,17,32,37]

Prolonged static lumbar loading periods may also have a significant impact on spinal stability. Spinal stability refers to the functional and mechanical integrity of its various structures (intervertebral joint, discs, ligaments, facet joint and its capsule, nerve roots, spinal cord, etc.) within their respective physiological ranges. Poor stability or a deficit in maintaining the various structures within their correct alignment during movement may result in excessive movement and lead to injury (prolapsed disc, facet impingement, nerve root compression, stenosis, etc.) and associated neurological implications of pain, impairment of movement, and substantial loss of work days. Two major components preserve spinal stability. The passive component consists of the var-

ious viscoelastic structures (ligaments, discs, dorso-lumbar fascia, facet capsules, etc.), and the active component consists of the musculature and its motor control.[1,23,24,27,35] Biomechanical testing confirmed that the viscoelastic tissues alone cannot provide sufficient stability to the spine across wide-ranging physiological loads and functions.[18] It is commonly accepted, therefore, that the musculature is the primary component that preserves stability under most circumstances in the physiological range.[23]

Since the active and passive stabilizers are inter-active due to their multi-feedback neurological closed loop control configuration, a synergistic relationship exists between them.[23,36,38,39] The load sharing between ligaments and muscles[20] is one such synergistic feedback concept, and the ligamento-muscular reflexive feedback[41,43] and facet capsule-muscular reflex feedback[12,13] are two other such concepts.

Panjabi[24] noted that small perturbations of the intervertebral joint about the neutral position are inherently stable and do not engage the viscoelastic tissues, whereas larger displacements meet with increased resistance from these structures. He defined the small perturbations within which the passive tissues are not engaged and the spine is inherently stable as neutral zones (NZ). Recently, the neuro-muscular neutral zone (NNZ) was described.[4,39] The NNZ was defined by the tension or displacement recorded in the lumbar spine at the instant that reflexive electromyogram (EMG) activity was first triggered in the multifidi muscles during flexion (or stretch). Similarly, during extension the lumbar tension and displacement recorded at the instant the EMG activity ceased was designated as the offset (or relaxation) NNZ. Under such circumstances, small NNZ are indicative of increased spinal stability, whereas larger or increased NNZ point to a deficient or decreasing stability. To date, it has been shown that the onset NNZ are significantly smaller than the respective offset NNZ. It was also shown that flexion–extension performed at faster rates resulted in a substantial decrease in the NNZ and in higher peak EMG, thus attesting to the increased stabilizing contribution from the musculature in fast movement.

Since static occupational activities were shown to result in laxity of the lumbar viscoelastic tissues and modification of the paraspinal musculature activity, one can expect that changes in stability would be present as well.[47] Indeed, clinicians who deal with occupational spine injuries note the common complaint of patients that a full work day was completed uneventfully, but while they were changing shoes/clothing in the locker room immediately after work, something "popped" in their back and triggered pain and disability. It is of value to assess if lumbar stability is impaired after prolonged static work while handling various load magnitudes.

The objective of this report is to assess the impact of various static load magnitudes on the NNZ and lumbar stability. We hypothesize that changing the magnitude of a sequence of static loads from mild to high, but within the physiological range, will have a significant impact on the NNZ and stability. Mild loads are expected to elicit small increases in the NNZ together with small transient decreases in muscular activity, whereas high-magnitude loads are expected to show a significant and prolonged increase in NNZ together with significant decreases in muscular activity. Lumbar stability is expected to be highly compromised by high loads in the 2–3 h immediately following physical activity.

It is expected that such new insight will expand our understanding of the motor control of spine stability, and injury mechanism, and it may provide a scientific basis for the design of static work schedules that may prevent instability or preventive measures to be employed immediately after work hours.

MATERIALS AND METHODS

Preparation. Nineteen adult cats, separated into two groups ($n = 10$, weight 3.54 ± 0.73 kg; and $n = 9$, weight 2.78 ± 0.40 kg), were used in this study. The sample size was determined by power analysis to be $n = 8$. Postanalysis, two additional preparations were added to the light load group to ensure that a lack of significance was independent of the sample size. Based on our long experience, weight is not a factor that influences responses, but the physical activity prior to testing is. Based on that, animals were stimulated to play and freely roam in a large room. The cats were anesthetized with 60 mg/kg chloralose, according to a protocol approved by the Institutional Animal Care and Use Committee. A superficial skin incision was made over the lumbar spine to expose the dorso-lumbar fascia, and an S-shaped stainless steel hook made of 1.5-mm-diameter rod was inserted around the supraspinous ligament between L_4 and L_5. The preparation was then positioned in a rigid stainless steel frame and fixed for subsequent EMG electrode insertion. The lumbar spine was isolated by means of two external fixators applied to the L_1 and L_7 posterior processes, respectively. The external fixation was intended to isolate the elicited flexion to the lumbar spine and prevent interaction with the thoracic, sacral, and
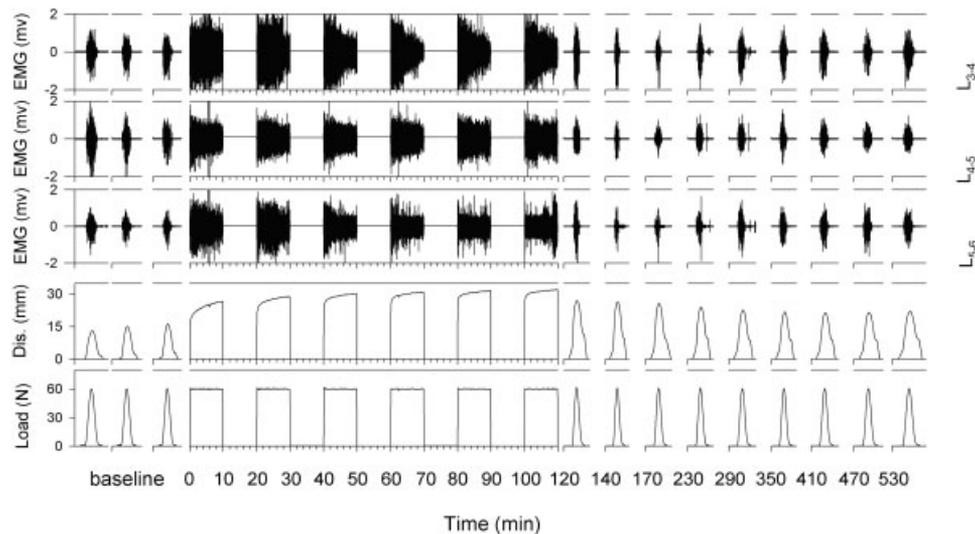
FIGURE 1. A typical recording of intramuscular EMG from the multifidi of the L-3/4 to L-5/6 levels in the top three traces with the displacement (fourth trace from top) during flexion and extension associated with the 60N load (bottom trace).

pelvic structures. A schematic of the set-up was provided in earlier reports.[5,41]

Instrumentation. Three pairs of fine stainless steel wire EMG electrodes (interelectrode distance: 3–4 mm) were inserted in the right L_{3-4}, L_{4-5}, and L_{5-6} multifidus muscles 6–8 mm lateral to the posterior spinous processes. A ground electrode was inserted into the gluteus muscle. Electrode locations were confirmed with dissection at the end of the experiment. EMG crosstalk from nearby muscles was not expected.[42] Each electrode pair constituted the input to a differential EMG amplifier with a 110-dB common mode rejection ratio, a gain of 500, and a bandpass filter in the range of 6–500 Hz. The EMG was sampled at 1,000 Hz before storage on a computer, and it was continuously monitored on an oscilloscope. The S-shaped stainless steel hook inserted around the L_{4-5} supraspinous ligament was connected to the crosshead of the Bionix 858 Material Testing System (MTS, Minneapolis, Minnesota). The load was applied through the MTS actuator with a computer-controlled loading system. Vertical displacement and tension applied to the hook around the supraspinous ligament were continuously monitored and sampled at 1,000 Hz before storage on a computer.

Protocol. Just prior to each test cycle or static load, a pretension of 1N was applied to standardize the baseline tension across all preparations. First, the baseline NNZs were established using three prestatic loading test cycles of 20N peak load at 0.25 Hz. The test cycles were applied with a 10-min rest (no load) between each. A peak load of 20N was selected, as it represents a mild load in the physiological range as determined in previous research.[29,41] Next, a static loading period was applied that consisted of six 10-min static loads at 20N, each separated by 10-min periods of rest, resulting in a cumulative static loading period of 60 min. A recovery period after static loading followed and consisted of a 7-h rest (no load). Single test cycles identical to those before the static loading session (20N peak @ 0.25 Hz) were applied at 10 min, 30 min, and 60 min, and then once every hour during the recovery period. Overall, nine test cycles were applied during the 7-h recovery, as shown graphically in the bottom panel of Figure 1.

In order to assess the NNZ response to high load magnitude, the same protocol described above was repeated with a static load of 60N, which was found to be near the high end of the physiological range.[29,41] For this protocol, the pre- and postloading test cycles were at 60N peak @ 0.25 Hz. Each of the two load magnitudes was applied only to one group of feline preparations. For the 20N load $n = 10$ and for the 60N load $n = 9$.

Data Processing. The NNZ analysis considered the recorded EMG, displacement, and tension applied
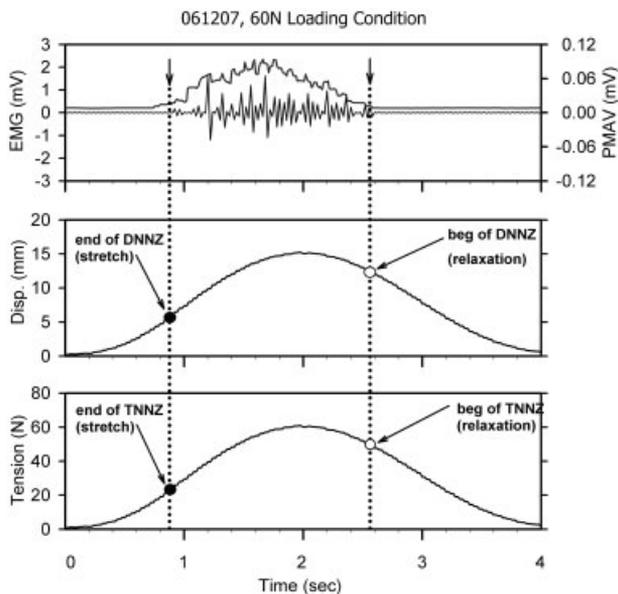
FIGURE 2. The graphical definition of the displacement neuromuscular neutral zones (DNNZ) and the tension neuromuscular neutral zones (TNNZ) associated with the multifidus EMG of a single flexion–extension cycle.

to the supraspinous ligament during the three test cycles prior to the static loading session and the nine test cycles during the 7-h recovery period. Each test cycle was analyzed in a 5-s window that consisted of 0.5 s before and 0.5 s after the 4-s cycle (0.25 Hz) as shown in Figure 2. The EMG signal was conditioned in the following manner: the EMG recorded from each lumbar level was full-wave-rectified, and a moving window of 200 ms was smoothed with a 200 ms lowpass filter. This was followed by similar smoothing of the next 200 ms window moved up 10 sampling points along the time axis, thereby obtaining the mean absolute value (MAV) of each EMG channel without creating a time delay. The MAV of the first 500 ms of each 5-s window, which occurred before the loading was initiated, was used as the baseline MAV. After tension was applied, muscle activation onset was considered to be when the MAV exceeded three times the baseline value. The three times baseline value was chosen in order to avoid false identification of NNZ due to spontaneous action potentials, which are common in resting EMG. The corresponding displacement and tension values during the stretch phase (increasing tension) of the cycle were recorded as the onset displacement neuromuscular neutral zone (DNNZ) and the onset tension neuromuscular neutral zone (TNNZ), respectively. When the EMG signal dropped below three times the baseline MAV during the relaxation (decreasing tension) phase of the cycle, the correspond-

ing displacement and tension were recorded as the offset DNNZ and TNNZ, respectively. This procedure was visually supervised to ensure that signal artifacts were not detected as thresholds.

The maximum MAV from each loading cycle was denoted as the peak MAV. The peak MAV from the three prestatic loading cycles were averaged together and used to normalize the peak MAV values in the cycles of the same preparation during the 7-h recovery period.

It was expected that any significant increase/decrease in peak MAV will indicate corresponding increase or decrease in overall muscular activity.

Statistics. The three initial preloading trials were averaged together to establish a more robust initial condition for statistical comparison. Statistical tests were applied to each static loading condition (20N, 60N) independently. Since multifidus segments are innervated from single ventral roots, each lumbar level (L_{3-4}, L_{4-5}, L_{5-6}) was independently assessed. Prior to analyses, each independent variable was visually inspected for normality, and an appropriate data transformation was applied if necessary. Two-way repeated-measures factorial analyses of variance (ANOVAs) were used to examine the effects of loading phase and time on the NNZs at each of the lumbar levels. Independent variables in this test included loading phase (stretch, relaxation) and time (baseline, recovery times); dependent variables included DNNZ and TNNZ. One-way repeated-measures ANOVAs were used to examine the effect of time on the remaining dependent variables. The independent variable in this test was time (baseline, recovery times); dependent variables included the peak MAV (PMAV). Upon significant interaction or main effect, pairwise comparisons were performed using Student's t-tests. The level of significance was established as $P = 0.05$.

A square root transformation was applied to TNNZ data from the 60N loading condition, and a log transformation was applied to PMAV data from both loading conditions to obtain a normal distribution. All figures presented are based on untransformed data.

Modeling. The mean ± standard deviation (SD) values of the DNNZ, TNNZ, and peak MAV during recovery for each lumbar level were fit with exponential-based models, as they represent the classical response of viscoelastic tissues.[40]

The time-course of the DNNZ thresholds during the stretch phase and relaxation phase of the test cycles during the recovery period were described by:

$$DISP(t) = D_0 + D_L(e^{-\tau_R/\tau_1}) \quad \{t|120 \leq t \leq 530\} \quad (1)$$

where D_0 is the intercept of the displacement (mm); D_L is the amplitude of exponential decay (mm); τ_1 is the exponential time-constant (min); τ_R is the time of the first recovery measurement relative to the beginning of the work-rest period (120 min).

The model was developed for the times 120 min to 530 min, which constitutes the 7 h of recovery poststatic loading.

The time-course of the TNNZ thresholds during the stretch phase and relaxation phase of the test cycles during the recovery period is described by:

$$TEN(t) = T_0 + (t - \tau_R)T_L(e^{-\tau_R/\tau_2})$$
$$+ T_M(e^{-\tau_R/\tau_3}) \quad \{t|120 \leq t \geq 530\} \quad (2)$$

where T_0 is the intercept of the tension (N); T_L affects the rise amplitude (N/sec); T_M is the amplitude of the decay dominating the end of the recovery period (N); $(t - \tau_R)T_L(e^{t-\tau_R/\tau_2})$ allows for a transient rise at the beginning of the recovery period; τ_2 affects the rates of rise and fall (sec); τ_3 is the exponential time-constant of the decay that dominates the end of the recovery period (min); τ_r is the time of the first recovery measurement (120 min).

The time-course of the peak MAV during the recovery period is described by:

$$peakMAV(t) = P_0 + P_L(e^{-\tau_r/\tau_4}) + P_M(1 - e^{-\tau_r/\tau_5})$$
$$+ (t - t_d)P_H(e^{-\tau_d/\tau_6}) \quad \{t|120 \leq t \leq 560\} \quad (3)$$

where P_0 is the intercept of the peak MAV (mm); P_L is the amplitude of the exponential decay (mm); P_M is the amplitude of the exponential increase (mm); t is time measured since the beginning of the experiment (min); τ_4, τ_5 are exponential time-constants (min); τ_r is the time of the first recovery measurement (120 min); $(t - \tau_d)P_He^{t-\tau_d/\tau_6}$ is the hyperexcitability term. This term has a delayed onset during the recovery period and is equal to zero when $t < \tau_d$.

Levenberg–Marquardt nonlinear regression algorithms were used to generate the best fit models, optimizing for the regression coefficient.

RESULTS

A typical recording of load, displacement, and EMG from the three lumbar levels for a 60N static load is shown in Figure 1.

Figure 2 provides the load, displacement, and EMG of the multifidi of a single lumbar level subjected to a test cycle, delineating the definition of the DNNZ and TNNZ from the projection of the initiation and cessation of the EMG during a loading cycle.

Displacement Neuromuscular Neutral Zones (DNNZ). Figure 3 displays the mean ± SD of the DNNZ for each loading condition (20N, 60N) before and after static loading.

There were no phase (stretch and release) × time interactions for any of the lumbar levels in the 20N condition ($P = 0.862$, $P = 0.818$, $P = 0.694$). Each level demonstrated significant main effects of phase ($P < 0.001$ for all levels) and time ($P < 0.001$ for all levels). Baseline values for the stretch phase were 2.8, 2.1, and 2.8 mm for L_{3-4}, L_{4-5}, L_{5-6} levels, respectively. These values increased by 2.4 to 3.2 times when measured immediately after the 1 h of static loading at 20N.

Baseline DNNZ values during extension were 4.5, 4.5, and 4.7 mm, respectively, and they increased by 1.7 to 2.0 times when measured after 20N static loading. DNNZs remained significantly elevated more than 5 h following static loading, and they decreased to near baseline at the seventh hour with values of 3.0, 2.9, and 2.8 mm (stretch phase) and 4.6, 5.4, and 5.3 mm (relaxation phase).

For the 60N loading condition, there were no phase × time interactions for any of the lumbar levels ($P = 0.450$, $P = 0.863$, $P = 0.591$), and all levels demonstrated significant main effects of phase ($P < 0.001$ for all levels) and time ($P < 0.001$ for all levels). Baseline values for the stretch phase were 5.9, 4.8, and 5.8 mm for L_{3-4}, L_{4-5}, L_{5-6} levels. These values increased by 1.6 to 1.9 times when measured after 60N static loading. Baseline values for extension were 9.2, 9.0, and 9.7 mm, respectively, and they increased by 1.6 to 1.7 times when measured after 60N static loading. DNNZs for flexion remained significantly elevated for more than 3 h following static loading and then decreased to below baseline at 3.4, 3.2, and 3.5 mm (stretch phase) and to near baseline at 9.6, 9.7, and 9.1 mm (relaxation phase) by the end of the recovery period.

Tension Neuromuscular Neutral Zones (TNNZ). Figure 4 displays the mean ± SD of the TNNZ for each of the two load magnitudes (20N, 60N) before and after static loading.

There were no phase × time interactions for any of the lumbar levels in the 20N condition ($P = 0.715$, $P = 0.919$, $P = 0.934$). Each level demonstrated significant main effects of phase ($P < 0.001$ for all levels) and time ($P < 0.001$ for all levels). Baseline values for the TNNZ in the stretch phase were 9.1,
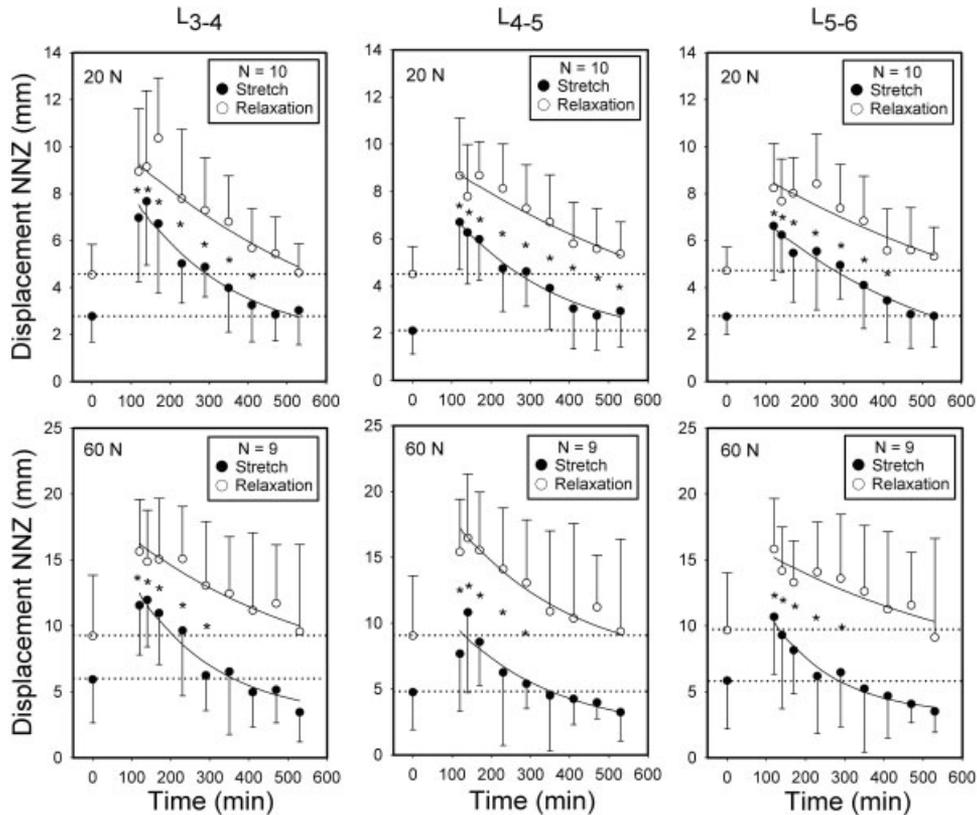
FIGURE 3. Mean (SD) of the displacement neuromuscular neutral zones (DNNZ) for the 20N (top row) and 60N (bottom row) loading conditions. Asterisks indicate a significant change of the DNNZ for both phases (stretch and relaxation) when compared to the baseline values. A decaying exponential was fit to each dataset and is displayed on each plot.

6.3, and 8.6 N for L_{3-4}, L_{4-5}, L_{5-6} levels, respectively. These values increased by 1.2 to 1.7 times within 30 min following 20N static loading. Baseline TNNZs in the relaxation phase were 12.2, 11.2, and 12.9 N, respectively, and they increased by 1.3 to 1.4 times within 1 h following 20N static loading. TNNZs remained significantly elevated through 2 h, 3 h, and 2 h for the three lumbar levels, respectively. They then decreased below baseline after reaching 6.7, 6.1, and 5.7 N (stretch phase) and 9.4, 11.7, and 12 N (relaxation phase) by the end of the 7-h recovery period.

There were no phase × time interactions in the 60N loading condition for any of the lumbar levels ($P = 0.586$, $P = 0.776$, $P = 0.560$), and all levels demonstrated significant main effects of phase ($P < 0.001$ for all levels) and time ($P < 0.001$ for all levels). Baseline TNNZ values for the stretch phase were 15.1, 12.1, and 15.4 N for the L_{3-4}, L_{4-5}, and L_{5-6} levels, respectively. Baseline values for the relaxation phase were 25.1, 28.0, and 32.6 N for the L_{3-4}, L_{4-5}, and L_{5-6} levels, respectively.

The L_{3-4} and L_{4-5} levels demonstrated significant increases of 1.2 and 1.7 times (stretch) and 1.2 and

1.7 times (relaxation) above baseline within the first hour following static loading. TNNZs of the L_{5-6} level did not increase following static loading. However, TNNZs decreased below baseline after the first hour of recovery for the L-3/4 level, after 30 min for the L-4/5 level and immediately after the loading for the L-5/6 level. All reached significance in the last 3–5 h of the 7-h recovery period.

For each lumbar level, both the DNNZs and TNNZs during the stretch phase were significantly smaller than their counterpart during the relaxation phase of loading. Differences between the loading phases have been discussed in detail in a previous publication.[39]

EMG Peak Mean Absolute Value (PMAV). Figure 5 displays the mean ± SD of the PMAV for each of the two load magnitudes (20N, 60N) before and after static loading.

In the 20N loading condition, significant changes with time were found in the L_{4-5} ($P < 0.001$) and L_{5-6} ($P < 0.001$) levels, while L_{3-4} did not vary significantly with time ($P = 0.122$). Pairwise comparisons revealed significant decreases of 56% and 78%
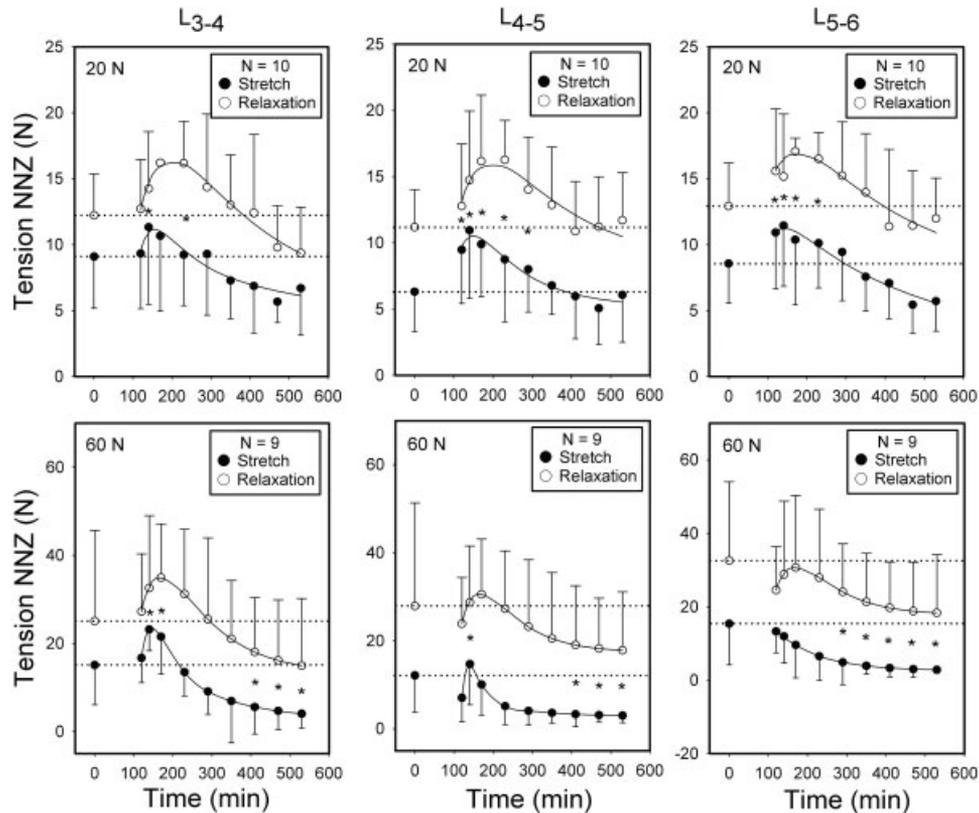
FIGURE 4. Mean (SD) of the tension neuromuscular neutral zones (TNNZ) for the 20N (top row) and 60N (bottom row) loading conditions. Asterisks indicate a significant change of the TNNZ for both phases (stretch and relaxation) when compared to the baseline values. An exponential equation with a transient rise was fit to each dataset and is displayed on each plot.

below the baseline values within the first hour of recovery for the L_{4-5} and L_{5-6} levels, respectively. Following this decrease there was a gradual but insignificant increase in the mean values that reached 42% and 39% above the baseline at the end of the seventh hour rest.

In the 60N loading condition, significant changes with time were found at all lumbar levels ($P = 0.050$, $P = 0.016$, $P < 0.001$). Pairwise comparisons revealed a significant decrease below baseline values within the first 2 h, 30 min, and 1 h for each of the three lumbar levels, respectively. The PMAV of the EMG increased above the baseline 1–2 h into the recovery for the three levels and reached significance in the last 3 h of recovery in the L-5/6 level. Increases of 30%–80% above baseline were reached by the end of the recovery period.

Models. Empirical models optimized to fit the DNNZ, TNNZ, and PMAV data are superimposed on Figures 3, 4, and 5. The regression coefficient (r^2) ranged from 0.848 to 0.978 for the DNNZ and from 0.828 to 0.945 for the TNNZ in the 20N loading condition. The regression coefficient ranged from

0.873 to 0.973 for the DNNZ and from 0.810 to 0.994 for the TNNZ in the 60N loading condition. The regression coefficient for the PMAV ranged from 0.455 to 0.975 and 0.584 to 0.870 for the 20N and 60N loading conditions, respectively. Regression coefficients are displayed in Table 1, and optimized parameters are displayed in Tables 2, 3 and 4..

DISCUSSION

The primary findings of this investigation consist of the following three issues.

Exposure of the lumbar spine to prolonged static loads, regardless of whether they are of low or high magnitude, elicits significant laxity in the viscoelastic tissues, which require up to 7 h of rest to recover to near baseline. During the postwork period the function of the viscoelastic tissues is compromised and manifests as a significant decrease in stability.

A neuromuscular compensation mechanism, however, is triggered some time after the work is completed and significantly reduces the instability. The neuromuscular compensation is sensitive to the
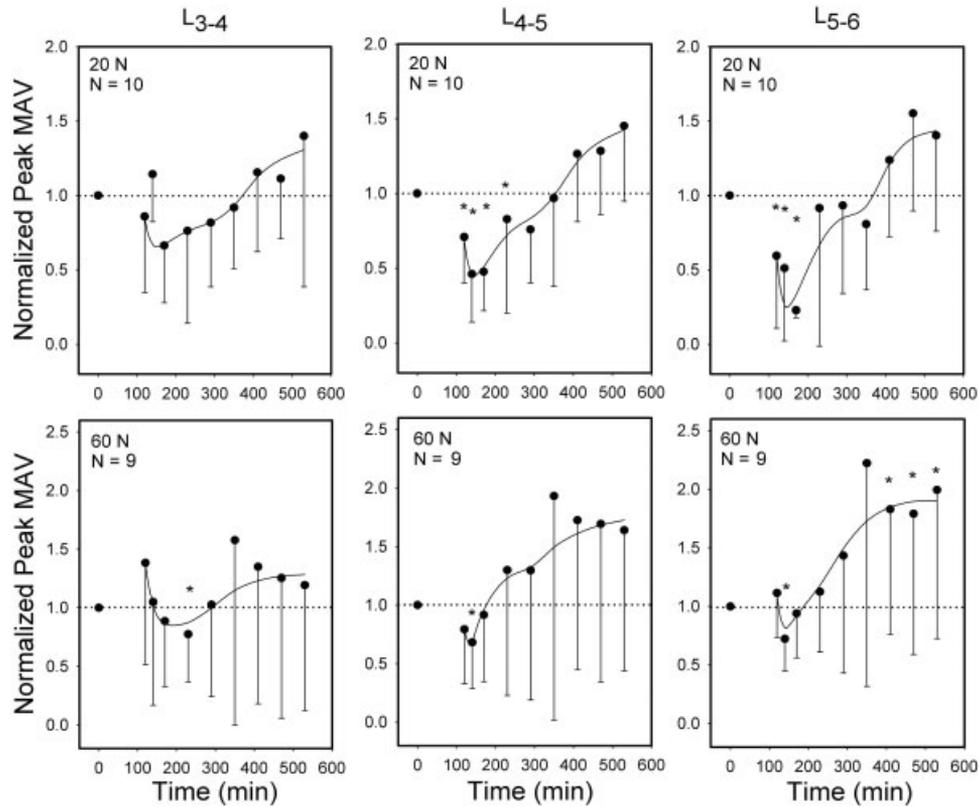
FIGURE 5. Mean (SD) of the peak mean absolute value (PMAV) of the EMG for the 20N (top row) and 60N (bottom row) loading conditions. All data were normalized within subjects to the baseline values. Asterisks indicate a significant change of the PMAV when compared to the baseline values.

load magnitude. It has a minor impact after prolonged static work at low load magnitudes and a very powerful impact after work at high loads. A direct relationship seems to exist between the load magnitude applied during the work period and the intensity of the neuromuscular compensation.

The delay associated with triggering of the compensation after work is also a factor. The compensation delay is inversely related to the load magnitude. Low loads trigger neuromuscular compensation nearly 3 h after the work is completed, whereas high load magnitudes trigger muscular responses immediately or within an hour postwork.

The DNNZ during the lumbar flexion phase was 2–3 times larger immediately after the static work at 20N as compared to 1.5–2 times larger after the same duration of work at 60N load. However, the DNNZ required nearly the full 7 h of rest to return to near baseline for the 20N load group, whereas the DNNZ corresponding to the 60N group returned to baseline by the third hour. In fact, for the 60N group, Figure 3 shows that the onset DNNZ continued to decrease below baseline, triggering muscular activity by the seventh hour of rest at displacement levels that were 33.3%–43% below baseline. While that may seem puzzling initially, it is fully explained by

Table 1. Regression coefficients (r^2) for the optimized DNNZ, TNNZ, and PMAV empirical equations.

Load	Phase	DNNZ			TNNZ			PMAV		
		L_{3-4}	L_{4-5}	L_{5-6}	L_{3-4}	L_{4-5}	L_{5-6}	L_{3-4}	L_{4-5}	L_{5-6}
20N	Stretch	0.957	0.978	0.967	0.945	0.845	0.969	0.455	0.975	0.880
	Relaxation	0.877	0.914	0.848	0.893	0.886	0.828			
60N	Stretch	0.916	0.953	0.958	0.974	0.994	0.988	0.584	0.722	0.870
	Relaxation	0.973	0.873	0.880	0.943	0.816	0.810			

Table 2. Optimized parameters for the empirical models describing behavior of the displacement neuromuscular neutral zones (DNNZ) versus time during the experiment.

$$DISP(t) = D_0 + D_L e^{[-(t - \tau_g)/\tau_1]}$$

Load	Phase	D_0	D_1	τ_1
		L_{3-4} Lumbar Level		
20N	Stretch	1.55	5.98	254
	Relaxation	0.02	9.27	636
60N	Stretch	3.00	9.47	208
	Relaxation	5.06	11.20	500
		L_{4-5} Lumbar Level		
20N	Stretch	1.54	5.16	272
	Relaxation	0.01	8.74	819
60N	Stretch	1.69	7.73	256
	Relaxation	6.37	10.87	301
		L_{5-6} Lumbar Level		
20N	Stretch	0.01	6.56	474
	Relaxation	0.02	8.46	902
60N	Stretch	3.23	7.10	164
	Relaxation	5.10	10.1	619

the pattern of the TNNZ and PMAV during that period. The TNNZ of the 60N group demonstrated a significant and fast decrease to below baseline within the first hour after the static work. The PMAV of the EMG also demonstrates that past the first hour after the static work, the musculature was active at significantly higher intensity than baseline. One can conclude that, as the neuromuscular compensation for the laxity in the viscoelastic tissues triggered the multifidi muscles earlier and at a much higher force, it increases the stiffness of the lumbar spine and

resists displacement. The increased stiffness of the spine, therefore, explains the decrease of the DNNZ to below baseline in the group loaded with 60N, as the spine offered increased resistance to the applied load. It is important to note, therefore, that the displacement measured not only represents the laxity of the viscoelastic tissues but also the level of activation of the musculature. Overall, the compensatory neuromuscular activation seems to be directly related to the load magnitude handled during the work session. Low loads triggered low magnitude compensation, whereas high loads produced a significant muscular response.

The compensatory response postwork at 20N load was very slow to trigger, requiring 3–4 h postwork (see Fig. 4, top). In contrast, the compensatory response to work at high loads triggered compensatory activity almost immediately (Fig. 4, bottom, L-5/6) and/or within an hour postwork (see Fig. 4, bottom, L-3/4 and L-4/5). Therefore, an inverse relationship exists between the postwork delay before the neuromuscular compensation triggers and the load handled during the work. High loads trigger compensatory neuromuscular responses early, whereas low loads manifest with longer delay to activate responses.

The source of the neuromuscular compensation postwork is of interest. Since the viscoelastic tissues became lax during the work period, one can conclude that the ligamento-muscular[41,43] and facet capsule–muscular[12,13] reflexes are significantly attenuated and nonfunctional.[35] With these sources eliminated as a mechanism, one has to consider the

Table 3. Optimized parameters for the empirical models describing behavior of the tension neuromuscular neutral zones (TNNZ) versus time during the experiment.

$$TEN(t) = T_0 + (t - \tau_R)T_L e^{[-(t - \tau_R)/\tau_2]} + T_M e^{[-(t - \tau_R)/\tau_3]}$$

Load	Phase	T_0	T_L	T_M	τ_2	τ_3
			L_{3-4} Lumbar Level			
20N	Stretch	3.71	0.12	5.82	48	465
	Relaxation	0.01	0.13	12.64	101	999
60N	Stretch	2.243	0.72	14.45	33	196
	Relaxation	12.55	0.45	14.69	69	200
			L_{4-5} Lumbar Level			
20N	Stretch	4.97	0.09	4.71	54	189
	Relaxation	7.90	0.11	5.15	100	399
60N	Stretch	2.79	1.16	4.25	19	136
	Relaxation	17.29	0.40	6.57	59	140
			L_{5-6} Lumbar Level			
20N	Stretch	0.01	0.04	11.01	60	597
	Relaxation	6.36	0.07	9.25	100	500
60N	Stretch	2.54	0.06	10.7	18	111
	Relaxation	17.81	0.36	6.71	65	120

Table 4. Optimized parameters for the empirical models describing behavior of the peak mean absolute value (PMAV) versus time during the experiment.

$$peakMAV(t) = P_0 + P_L e^{[-(t-\tau_t)/\tau_L]} + P_M(1 - e^{[-(t-\tau_t)/\tau_5]}) + (t-\tau_d)P_H e^{[-(t-\tau_d)/\tau_6]}$$

Load	P_0	P_L	P_M	τ_4	τ_5	P_H	τ_6	τ_d
L_3 Lumbar Level								
20N	0.52	0.34	0.39	16	70	0.004	302	328
60N	0.38	1.00	0.73	33	110	0.002	230	270
L_{4-5} Lumbar Level								
20N	−0.05	0.75	0.97	20	70	0.005	302	328
60N	0.38	0.42	1.00	5	57	0.005	198	295
L_{5-6} Lumbar Level								
20N	−0.56	1.18	1.55	21	71	0.010	131	362
60N	0.47	0.62	1.00	14	88	0.005	240	250

tissue damage to be a potential source. The neuromuscular hyperactivity and high stiffness of the lumbar spine in patients with confirmed low back disorders is well known.[5,8,10,21,28,33,44] If one considers that work at high loads develops higher laxity in the viscoelastic tissues and therefore more microdamage in the collagen tissues[46] and the associated acute inflammatory response,[16,38] then it is clear that some tissue damage and a disorder[38] was developed during the work duration. Such microdamage may be proportional to the load level, being higher for higher loads. Putting these issues in perspective, one can reasonably conclude that the potential event that triggers the neuromuscular compensation of spinal stability is the level of microdamage developed in the viscoelastic tissues. Work while handling low loads was shown to be a relatively low or no-risk activity, and it is probably associated with minimal microdamage to the tissues.[29] The required neuromuscular compensatory response to such conditions is low and slow to trigger. Conversely, work at high loads results in pronounced microdamage in the viscoelastic tissues and that, in turn, triggers a fast and pronounced neuromuscular response. That the compensatory response of the musculature with strong contraction shields the damaged tissue from further exposure and maintains or restores the compromised stability in this case is a reasonable conclusion.

Amazingly, and contrary to our hypothesis, work while handling low loads leaves the spine without viscoelastic or muscular support and therefore unstable and at high risk of instability and injury for over 3 times longer than work at high loads. The slow onset compensation is also of low magnitude. Work while subjected to low loads, therefore, is not a risk factor for cumulative low back disorder, but it may not be excluded as a no-risk occupational activity. It exposes the lumbar spine to instability for 3–4 h postwork. One should note that two separate disorders are engaged with this occupational activity. They are cumulative low back disorder arising from soft tissue damage/inflammation and an instability disorder arising from laxity and deficit in muscular function. One disorder does not imply the presence of the other—in fact, one should expect only one of them to be present at any given time.

Workers and athletes with lumbar injury or low back pain who are seen in spine clinics commonly describe the uneventful engagement in prolonged activity over the day. Yet at the end of the activity, while they are changing clothes, a simple movement such as attempting to put on shoes or turning to pick up their lunch box results in a distinct "pop" in the spine followed by pain. While such descriptions are common, they are so far anecdotal. The data reported in this study experimentally confirm that a significant deficit in passive and active stability is present in the hours immediately after a prolonged work period, and it is not surprising or even expected that even minor/routine movements may result in instability and associated injury. It is imperative that workers and athletes find means to protect their lumbar spine in this period as a measure of prevention.

The data obtained in this research was from the feline and requires qualification before assessing its applicability to humans. The feline model is considered a classical preparation in neurophysiology and especially in neuromuscular applications. The validity of such a model in human neurophysiology was repeatedly established as far as nerves, muscles, reflexes, innervation, etc. The biomechanical responses of the feline to loads, directions of motion, and soft tissues (ligaments, capsules, discs, etc.) were also explored. Smit[34] identified the mechanical char-

acteristics of quadruped spines and compared them with that of humans. He concluded that the quadruped spine can serve as a valuable model to study the biomechanics of the human spine. Recently, Iannuzi et al.[11] compared the cat spine with the human spine in assessing torque limits and motion angles. They also concluded that within the conditions tested "the similar ranges of motion measured in the cat and human lumbar spines support the use of the cat as a model to study neuromechanics of the lumbar spine." Our own work[45] identified identical physiological strain ranges of lumbar ligaments to that of humans.[22] Excluding scaling and "industrial history," the similarity of the cat's neuromuscular system and lumbar mechanical behavior allows a certain liberty in expressing some conclusions from feline experiments to be applicable to humans. Finally, and importantly, experiments with human subjects consistently verify the various neuromuscular and mechanical responses observed in the in vivo feline under cyclic and static work, including the development of laxity and neuromuscular disorders.[3,6,7,14,17,32,37]

In conclusion, regardless of the load magnitude applied during static work, the first 1–3 h after completing the work finds the lumbar spine without the stabilizing function of the ligaments and simultaneously with deficient protection from the muscles. This immediate postwork period should be considered a high-risk window for instability and potential injury. In fact, prolonged work at low loads does not benefit from neuromuscular compensation as fast and as powerfully as in postwork at high loads and should be attended to with extra care.

REFERENCES

1. Adams M. Re: spine stability: the six blind men and the elephant. Clin Biomech (Bristol, Avon) 2007;22:486; author reply 7–8.
2. Courville A, Sbriccoli P, Zhou BH, et al. Short rest periods after static lumbar flexion are a risk factor for cumulative low back disorder. J Electromyogr Kinesiol 2005;15:37–52.
3. Dickey J, McNorton S, Potvin J. Repeated spinal flexion modulates the flexion-relaxation phenomenon. Clin Biomech 2003;18:783–789.
4. Eversull E, Solomonow M, Zhou BH, et al. Neuromuscular neutral zones sensitivity to lumbar displacement rate. Clin Biomech (Bristol, Avon) 2001;16:102–113.
5. Fisher A, Chang C. Electromyographic evidence of paraspinal muscles spasms during sleep in patients with low back pain. Clin J Pain 1985;1:147–154.
6. Granata KP, Marras WS. Cost-benefit of muscle co-contraction in protecting against spinal instability. Spine 2000;25:1398–1404.
7. Granata KP, Rogers E, Moorhouse K. Effects of static flexion-relaxation on paraspinal reflex behavior. Clin Biomech 2005;20:16–24.
8. Haig A, Weisman G, Haugh L, Pope M, Grobler L. Prospective evidence for change in paraspinal muscle activity after herniated nucleus pulposus. Spine 1993;18:926–930.
9. Hoogendoorn WE, Bongers PM, de Vet HC, et al. Flexion and rotation of the trunk and lifting at work are risk factors for low back pain: results of a prospective cohort study. Spine 2000;25:3087–3092.
10. Hoyt W, Hunt H, DePauw M, et al, EMG assessment of chronic low back pain syndrome. J Am Osteopath Assoc 1981;80:728–730.
11. Ianuzzi A, Pickar J, Khalsa P. Determination of torque limits for human and cat lumbar spine specimen during displacement controlled physiological motion. Spine J 2007 (to be published).
12. Indhal A, Kaigle A, Reikeras O, Holm S. Interaction between porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. Spine 1997;22:2834–2840.
13. Indhal A, Kaigle A, Reikgras O, Holm S. EMG response of the porcine multifidus musculature after nerve stimulation. Spine 1995;20:2652–2658.
14. Krajcarski S, Wells R. The time variation pattern of mechanical exposure and the reporting of low back pain. Theor Issues Ergon Sci 2006:1–27.
15. LaBry R, Sbriccoli P, Zhou BH, et al. Longer static flexion duration elicits a neuromuscular disorder in the lumbar spine. J Appl Physiol 2004;96:2005–2015.
16. Leadbetter W. An introduction to sports induced soft tissue inflammation. In: Leadbetter W, Buckwalter J, Gordon S, editors, Sports induced inflammation; clinical and basic science concepts. Park Ridge, IL: American Academy of Orthopedic Surgeons.
17. Little J, Khalsa, P. Human lumbar spine creep during cyclic and static flexion: creep rate, biomechanics, and facet joint capsule strain. Ann Biomed Eng 2005;33:391–401.
18. Lucas D, Bresler B. Stability of the ligamentous spine. Biomechanics Laboratory, UCSF/B. Report No. 40, 1961:1–41.
19. Marras W. Occupational low back disorder causation and control. Ergonomics 2000;43:880–902.
20. McGill S, Kippers V. Transfer of loads between lumbar tissues during the flexion-relaxation phenomenon. Spine 1994;19:2190–2196.
21. Miller D. Comparison of EMG activity in the lumbar paraspinal muscles of subjects with and without chronic low back pain. Phys Ther 1985;65:1347–1354.
22. Panjabi M, Goel V, Takata K. Physiologic strains in the lumbar spinal ligaments. Spine 1982;7:192–203.
23. Panjabi M. Clinical spinal stability and low back pain. J Electromyogr Kinesiol 2003;13:371–379.
24. Panjabi M. The stabilizing system of the spine. II. Neutral zone and instability hypothesis. J Spinal Disord 1992;5:390–397.
25. Punnett L, Fine L, Keyserling W, et al. Back disorders and nonneutral trunk postures of automobile assembly workers. Scand J Work Environ Health 1991;17:337–346.
26. Punnett L, Wegeman D. Work related musculoskeletal disorders. The epidemiologic evidence and the debate. J Electromyogr Kinesiol 2004;14:13–23.
27. Reeves N, Narendra K, Cholewicki J. Spine stability: the six blind men and the elephant. Clin Biomech (Bristol, Avon) 2007;22:266–274.
28. Roland M. Critical review of the evidence for a pain-spasm-pain cycle in spinal disorders. Clin Biomech 1986;1:102–109.
29. Sbriccoli P, Solomonow M, Zhou BH, et al. Static load magnitude is a risk factor in the development of cumulative low back disorder. Muscle Nerve 2004;29:300–308.
30. Sbriccoli P, Solomonow M, Zhou BH, et al. Work to rest durations ratios exceeding unity are a risk factor for low back disorder; a feline model. J Electromyogr Kinesiol 2007;17:142–152.

31. Sbriccoli P, Yousuf K, Kupershtein I, et al. Static load repetition is a risk factor in the development of lumbar cumulative musculoskeletal disorder. Spine 2004;29:2643–2653.

32. Shin G, Mirka G. An in vivo assessment of the low back response to prolonged-flexion. Interplay between active and passive tissues. Clin Biomech 2007;22:965–971.

33. Shivonen T, Partanen J, Hanninen O, Soimakallio S. Electric behavior of low back muscles during lumbar pelvic rhythm in low back pain and healthy controls. Arch Phys Med Rehab 1991;72:1080–1087.

34. Smit T. The uses of a quadruped as an in vivo model of the spine-biomechanical considerations. Eur Spine J 2002;11;137–144.

35. Solomonow M. Sensory-motor control of ligaments and associated neuromuscular disorders. J Electromyogr Kinesiol 2006;16;549–567.

36. Solomonow M. External control of the neuromuscular system. IEEE Trans Biomed Eng 1984;31:752–763.

37. Solomonow M, Baratta R, Banks A, Freudenberger C, Zhou B. Flexion-relaxation response to static lumbar flexion. Clin Biomech 2003;18:273–279.

38. Solomonow M, Baratta RV, Zhou B, Burger E, Zieske A, Gedalia A. Muscular dysfunction elicited by creep of lumbar viscoelastic tissues. J Electromyogr Kinesiol 2003;13:381–396.

39. Solomonow M, Eversull E, Zhou B, Baratta RV, Zhu M. Neuromuscular neutral zones associated with viscoelastic hysteresis during cyclic lumbar flexion. Spine 2001;26:E-314–324.

40. Solomonow M, Zhou B, Baratta R, Lu Y, Zhu M, Harris M. Biexponential recovery model of lumbar viscoelastic creep and reflexive muscular activity after prolonged cyclic loading. Clin Biomech 2000;15:167–175.

41. Solomonow M, Zhou B, Harris M, Lu Y, Baratta R. The ligamento-muscular stabilizing system of the spine. Spine 1998;23:2552–2562.

42. Solomonow M, Baratta R, Bernardi M, Zhou B, Lu Y, Acierno S. Surface and wire EMG crosstalk in neighboring muscles. J Electromyogr Kinesiol 1994;4:131–142.

43. Stubbs M, Harris M, Solomonow M, Zhou B, Lu Y, Baratta R. The ligamento-muscular protective reflex in the lumbar spine. J Electromyogr Kinesiol 1998;8:197–204.

44. van Dieen JH, Selen LP, Cholewicki J. Trunk muscle activation in low-back pain patients, an analysis of the literature. J Electromyogr Kinesiol 2003;13:333–351.

45. Williams M, Solomonow M, Zhou B, Baratta R, Harris M. Multifidus spasms elicited by prolonged lumbar flexion. Spine 2000;25:2916–2924.

46. Woo S, Apreleva M, Hoher J. Tissue mechanics of ligaments and tendons. In: Kumar S, editor. Biomechanics in ergonomics. London: Taylor & Francis; 1999.

47. Youssef J, Davidson B, Zhou B, Lu Y, Patel V, Solomonow M. Neuromuscular neutral zones response to static lumbar flexion; muscular stability compensator. Clin Biomechan 2008; 23:870–880.