Neuromuscular neutral zones response to cyclic lumbar flexion

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The in vivo lumbar spine of the anaesthetized feline was subjected to passive cyclic anterior flexion–extension at 0.25 Hz and 40 N peak load for cumulative 60 min duration. Displacement (or displacement neuromuscular neutral zones—DNNZ) and tension (or tension neuromuscular neutral zones—TNNZ) at which reflexive EMG activity from the multifidi muscles was initiated and terminated were recorded, for single-test cycles, before and for 7 h after cyclic loading. Displacement and tension NNZs increased significantly after loading. The displacement NNZs decreased exponentially to near baseline by the 7th hour of rest. The tension NNZs, however, decreased to below the baseline after the 2nd to 3rd hour after loading and continued decreasing into the 7th hour. Peak EMG significantly decreased (49–57%) to below the baseline immediately after loading and then exponentially increased, exceeding the baseline by the 2nd to 3rd hour and reaching 33–50% above baseline by the 7th hour. EMG median frequency decreased after loading and then exceeded the baseline after the 3rd hour, indicating initial de-recruitment, followed by recruitment of new motor units. These findings suggest that the lumbar spine was exposed to instability for 2–3 h after cyclic loading, due to concurrent laxity of the viscoelastic tissues and deficient muscular activity. A delayed neuromuscular compensation mechanism was found to exist, triggering the musculature significantly earlier and at higher magnitude than baseline, while the viscoelastic tissues were still lax. Thus, it is suggested that prolonged cyclic loading may compromise lumbar stability during the immediate 2–3 h post-loading, increasing the risk of injury.

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1. Introduction

Spinal stability is a complex issue, the full extent of which is not fully delineated (Adams, 2007). One component of the structural stability of the intervertebral joints is composed of the effects of the viscoelastic properties of the ligaments, discs and facet capsules. When vertebrae are displaced relative to each other, the various viscoelastic tissues deform and generate tension expressed as resistance to destabilizing motion. This passive property of the viscoelastic tissues is minimal for small perturbations about the neutral position and sharply increases for larger displacements. Panjabi (1996, 1992) designated the range of small perturbation where the viscoelastic tissues are minimally engaged as neutral zones (NZ) within which the spine is inherently stable.

Another important component of spine stability is the contribution of the muscles and their motor control (Panjabi, 1996; Reeves et al., 2007; Adams, 2007). Recently, (Eversull et al., 2001; Solomonow et al., 2001) described the neuromuscular neutral zones (NNZ), where passive intervertebral motion above a certain displacement or a load above a certain magnitude, triggers the reflexive activation of the musculature to preserve the stability (Stubbs et al., 1998; Solomonow et al., 1998). Under such definition, initiation of muscular activity at a higher displacement (Displacement NNZ) or higher tension (Tension NNZ) than normal is indicative of a decrease or deficiency in stability, as more motion is performed without the protection of the musculature. Conversely, decrease in the displacement or tension level, or lower displacement or tension NNZ, is indicative of increased stability. To date, it was shown that the NNZ during flexion are smaller than during extension and that the NNZ display a gradual decrease concurrent with EMG increase as the velocity of spinal flexion–extension increases.

Cyclic/repetitive sports and occupational activities were shown to trigger high rates of musculoskeletal disorders when performed over long periods (Marras, 2000; Silverstein et al., 1986; Punnett et al., 1991; Hoogendoorn et al., 2000). The epidemiology was recently confirmed biomechanically and physiologically in in vivo models (Navar et al., 2006; Le et al., 2007; Hoops et al., 2007) and in humans (Granata et al., 2005, 1999; Olson et al., 2004, 2006; in press; Shin and Mirka, 2007; Li et al., 2007; Dickey et al., 2003;
Karajcarsi and Wells, 2006). Prolonged periods of exposure to cyclic lumbar loading were shown to develop substantial laxity/creep in the viscoelastic tissues and in turn, significant changes in the activation pattern of the spinal musculature. A disorder consisting of spasms, temporary attenuation of muscle activity followed by hyperexcitability was observed. It is expected that cyclic activity of the lumbar spine may also elicit pronounced changes in the NNZ and elicit pronounced changes in spinal stability.

We hypothesize that 60 min of cyclic lumbar loading at a moderate load will cause significant enlargement of the NNZ, immediately after loading and that several hours of rest will be required to restore normal NNZ. We further predict that pronounced changes may be observed in the EMG amplitude and its motor control when comparing it before and after cyclic loading.

Such information can afford new insights into the changes in the motor control responsible for the stability of the lumbar spine after cyclic work, the potential for injury and development of a means for its prevention, as well as baseline data for design of safe work scheduling.

2. Methods

2.1. Preparation

Seven adult cats, with an average weight of 3.95 ± 0.37 kg, were used in this study. They were anesthetized with 60 mg/kg chloralose, according to a protocol approved by the Institutional Animal Care and Use Committee. A superficial skin incision overlaying the lumbar spine was made to expose the dorso-lumbar fascia, and an S-shaped stainless steel hook made of 1.5-mm-diameter rod was applied around the supraspinous ligament between L4 and L5. The preparation was then positioned in a rigid stainless steel frame and the lumbar spine was isolated by pelvic structures, but not to prevent any motion.

2.2. Instrumentation

Three pairs of stainless steel fine wire EMG electrodes (interelectrode distance: 3–4 mm) were inserted into the right L3–4, L4–5, and L5–6 multifidus muscles 6–8 mm laterally from the posterior spinal processes. A ground electrode was inserted into the gluteus muscle. 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. The EMG was recorded with a sampling rate of 1000 Hz, and it was continuously monitored on an oscilloscope. The S-shaped stainless steel hook inserted around the L4–5 supraspinous ligament was connected to the crosshead of the Bionix 858 material testing system (MTS, Minneapolis, MN), in which a load cell was located. The load was applied through the MTS actuator with a computer-controlled loading system and monitored continuously along with the vertical displacement of the actuator. The load cell and displacement outputs of the Bionix 858 MTS were also sampled into the computer at 1000 Hz, along with the EMG signals.

Under such loading condition, the lumbar spine underwent anterior flexion–extension while straining the supraspinous, intraspinous and posterior longitudinal ligaments as well as the ligamentum flavum, facet capsule and dorso-lumbar fascia. The discs in the lumbar spine were also deformed, expanding in their dorsal aspects while narrowing in their ventral aspects during the flexion phase. Overall, the neuromuscular response represents the reflexive activation of several lumbar viscoelastic components similar to those active during flexion–extension.

2.3. Protocol

A pre-load of 1 N was applied just prior to each single period of cyclic loading in order to produce a standard baseline across all preparations. Initially, single cycles of 40 N peak load at 0.25 Hz were applied with a 10 min rest at no load between each cycle. This phase was performed to establish the normal NNZ before cyclic loading. Following, a set of six 10 min cyclic loading periods at 40 N peak, each followed by 10 min rest was applied for a cumulative cyclic loading period of 60 min. The following recovery phase consisted of 7 h of rest at no load, during which single-test cycles of 40 N peak load at 0.25 Hz were applied. The single cycles were applied at 10, 30 and 60 min after the cyclic loading phase terminated, and then once at every hour. Overall, 9 test cycles were applied during the 7 h recovery period. Fig. 1 shows the schematic representation of the cyclic loading with the single-cycle tests before and after. EMG, load and displacement were recorded throughout the protocol.

2.4. Data analysis

The analysis considered the recorded EMG, vertical displacement and cyclic load applied to the supraspinous ligament during the three pre-loading cycles and the nine cycles during the recovery period. Each cycle was analyzed in a 5 s window including 0.5 s before and 0.5 s after the 4 s cycle (0.25 Hz). EMG threshold analysis was performed as follows: The first 500 ms of each EMG record, which was before the initiation of movement by the actuator, was used as a benchmark for baseline EMG signal level. The mean absolute value (MAV) of this baseline was calculated: the signal recorded from each lumbar level was full wave rectified and

\[
\text{ MAV } = \frac{1}{T} \int |x(t)| \, dt
\]

where T is the duration of the window. The MAV was used to determine the threshold of the signal, which was set at 1.5 MAV. EMG activities above the threshold were considered as active.

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\frac{1}{2} T \int |x(t)| \, dt > 1.5 \times \text{ MAV}
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smoothed with a 200 ms moving average filter (every 10 points) to yield the EMG MAV. The filter was centered on the window under consideration in order to prevent any time lags from affecting the data. After the initial 0.5 s period, any point along the absolute value (full wave rectified) of the EMG during the given cycle that exceeded three times the baseline MAV was denoted as the threshold of activity in that channel. The corresponding tension and displacement values during the stretch phase (increasing load) of the cycle were recorded as the tension neuromuscular neutral zone (TNNZ) and displacement neuromuscular neutral zone (DNNZ) DNNZ threshold values for that particular trial and lumbar level. Similarly, as the EMG dropped below the three times baseline MAV threshold in the release phase (decreasing load) of the cycle, the corresponding tension and displacement were recorded as the TNNZ and DNNZ thresholds of cessation of activity. This automatic procedure was visually supervised to ensure that any unexpected signal artifacts were not detected as thresholds. Furthermore, in cases where the EMG traces included spams along the baseline, or the EMG discharge during the flexion-extension was below three times the baseline [for example, see Fig. 2, L-3/4 level, 110–170 min interval], the thresholds were determined visually.

The DNNZ and TNNZ from corresponding cycles during the recovery period for each of the preparations were pooled across lumbar level, and the mean (± SD) were calculated and plotted as a function of time. The DNNZ and TNNZ of the three cycles applied before cyclic loading was initiated were averaged for each preparation to yield a mean and SD, and then pooled with those of the other preparations to use as the pre-loading NNZ baselines.

The peak MAV (PMAV) of the first three cycles recorded before cyclic loading was used as a normalization value for the PMAV recorded in the test cycles of the same preparation during the 7 h recovery period. A single-peak value was used as a normalization value for the PMAV recorded in the test cycles of the same preparation during the 7 h recovery period. A single-peak value was used as a normalization value for the PMAV recorded in the test cycles of the same preparation during the 7 h recovery period. A single-peak value was used as a normalization value for the PMAV recorded in the test cycles of the same preparation during the 7 h recovery period.

Since multifidus segments are not innervated from multiple roots (MacIntosh et al., 1986), the data was divided into three lumbar levels (L-3/4, L-4/5, L-5/6) for statistical analyses. The DNNZ, TNNZ and PMAV data were visually inspected for normality. If a distribution did not appear normal, an appropriate data transformation was applied. Two-way repeated measures ANOVAs were used to test for differences in the stretch and relaxation phases and changes over time of the DNNZ and TNNZ. The independent variables included time (pre-cyclic loading, recovery times) and loading phase (stretch, relaxation). All higher order factorial terms were included in the statistical models to test for interaction of the independent variables. One-way repeated measures ANOVAs were used to test for changes over time in the EMG-based variables. The independent variables included time (pre-cyclic loading, recovery times) and the dependent variables were normalized PMAV and median frequency. Upon determining a significant interaction or main effect, pair-wise comparisons were performed using a Student’s t-test. Level of significance for all tests was set as \( p = 0.05 \).

2.5. Statistics

Since multifidus segments are not innervated from multiple roots (MacIntosh et al., 1986), the data was divided into three lumbar levels (L-3/4, L-4/5, L-5/6) for statistical analyses. The DNNZ, TNNZ and PMAV data were visually inspected for normality. If a distribution did not appear normal, an appropriate data transformation was applied. Two-way repeated measures ANOVAs were used to test for differences in the stretch and relaxation phases and changes over time of the DNNZ and TNNZ. The independent variables included time (pre-cyclic loading, recovery times) and loading phase (stretch, relaxation). All higher order factorial terms were included in the statistical models to test for interaction of the independent variables. One-way repeated measures ANOVAs were used to test for changes over time in the EMG-based variables. The independent variables included time (pre-cyclic loading, recovery times) and the dependent variables were normalized PMAV and median frequency. Upon determining a significant interaction or main effect, pair-wise comparisons were performed using a Student’s t-test. Level of significance for all tests was set as \( p = 0.05 \).
where \( F_0 \) is the intercept of the peak MF (Hz); \( F_L \) is the amplitude of exponential decay dominating beginning of recovery period (Hz); \( F_M \) is the amplitude of exponential increase following decay in beginning of recovery period (Hz); \( t_r \) is the time of first recovery measurement (120 min); \( t_o \) is the exponential time constant of exponential decay dominating beginning of recovery period (min); \( t_d \) is the exponential time constant of exponential increase following decay in beginning of recovery period (min); \( t_8 \) is the exponential time constant of exponential decay dominating beginning of recovery period (min); \( t_9 \) is the exponential time constant of exponential increase following decay in beginning of recovery period; \( F_L(\exp(-t/t_{d})) \) Allows for exponential decay dominating beginning of recovery period; \( F_M(1/\exp(-t/t_{d})) \) Allows for exponential increase following decay in beginning of recovery period; \( (t/t_{d})F_H(\exp(-t/t_{d})) \) Allows for hyperexcitability term dominating end of recovery period (equal to zero when \( t < t_{d} \)).

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

3. Results

A typical recording of the raw EMG from L-3/4, L-4/5 and L-5/6, and the associated tension and displacement before, during and after the cyclic loading period is shown in Fig. 2. The same parameters corresponding to a typical single cycle are given in Fig. 3 with the arrows indicating the initiation and cessation of EMG, and the corresponding displacement and tension.

3.1. Displacement neuromuscular neutral zones (DNNZ)

Fig. 4 displays the mean ± SD of the DNNZ before and after the cyclic loading. The DNNZ demonstrated changes with time (\( P<0.001 \) for all levels) and also loading phase (\( P<0.001 \) for all levels), and no time × loading phase interaction (L-3/4: \( P = 0.990 \), L-4/5: \( P = 0.937 \), L-5/6: \( P = 0.863 \)). DNNZ for both phases remained significantly elevated through 4 h after cyclic loading for levels L-3/4 and L-4/5, and through 3 h after cyclic loading for level L-5/6 (see Fig. 4). The mean of the baseline DNNZ during the stretch phase were 2.7, 2.1 and 2.7 mm for the L-3/4, L-4/5 and L-5/6 lumbar levels, respectively. These values increased over threefold to 9.8, 9.2 and 9.3 mm immediately after the cyclic loading period. The final mean DNNZ at the end of the 7 h recovery period were 2.6, 2.8 and 2.5 mm for the three lumbar levels, respectively.

The mean of the baseline DNNZ during the relaxation phase were 6.1, 5.6 and 7.1 for the three lumbar levels, respectively. The DNNZ increase of roughly twofold to 12.7, 12.4 and 12.8 mm following the cyclic loading. A gradual decrease to baseline over the 7 h recovery was observed to final DNNZ of 6.3, 6.0 and 6.0 mm for the three lumbar levels, respectively.

For each lumbar level, the DNNZ during stretch were significantly smaller than their counterpart during relaxation. Differences between the loading phases were discussed in detail in a previous paper and will not be duplicated here (Solomonow et al., 2001).

3.2. Tension neuromuscular neutral zones (TNNZ)

The mean TNNZ before and after the cyclic loading is shown in Fig. 5. The TNNZ demonstrated changes with time (\( P<0.001 \) for all levels).
levels) and also loading phase ($P < 0.001$ for all levels), and no time × loading phase interaction (L-3/4: $P = 0.895$, L-4/5: $P = 0.984$, L-5/6: $P = 0.910$). TNNZ for both phases remained significantly elevated through 2 h after cyclic loading in level L-3/4, and through 1 h after cyclic loading in levels L-4/5 and L-5/6. The TNNZ significantly decreased to below baseline values in the 6th and 7th hours in level L-3/4, and in the 5th–7th hours in levels L-4/5 and L-5/6 (see Fig. 5). The mean of the baseline TNNZ during the stretch phase were 8.8, 7.0 and 9.1 N for the three lumbar levels, respectively. The TNNZ increased to 19.0, 15.6 and 16.9 N immediately after cyclic loading and gradually decreased to reach 3.3, 3.9 and 3.2 N at the end of the 7th hour of recovery.

The mean of the baseline TNNZ during the relaxation phase was 12.4, 11.1 and 18.3 N for the three lumbar levels, respectively. The TNNZ increased by nearly twofold to 25.7, 25.3 and 29.5 N following cyclic loading. The mean TNNZ gradually declined over the recovery period, reaching 8.4, 7.3 and 9.1 N at the end of the 7th hour of recovery.

For each lumbar level, the TNNZ during stretch were significantly smaller than their counterpart during relaxation. Differences between the loading phases were discussed in detail in a previous paper and will not be duplicated here (Solomonow et al., 2001).

3.3. EMG peak mean absolute value (PMAV)

Fig. 6 shows the normalized PMAV before and after the cyclic loading. The PMAV demonstrated a significant change over time for each lumbar level ($P < 0.0001$). A significant decrease of 49%, 57% and 51% was evident immediately after cyclic loading that remained below the baseline values for one hour during recovery (see Fig. 6). The PMAV exceeded baseline levels after the third hour for the L-3/4 level, about the second hour for the L-4/5 level, and just before the third hour for the L-5/6 level. The L-5/6 lumbar level was significantly higher in the 5th–7th hours during recovery. The PMAV reached 1.14, 1.45 and 1.59, or 14–59% above baseline, at the 7th hour.

3.4. EMG median frequency (MF)

Fig. 7 shows the MF before and after the cyclic loading. The MF demonstrated a significant change over time ($P < 0.0001$) for each
lumbar level. The MF decreased as the site of measurement moved down the lumbar levels (from L-3/4 to L-5/6). The baseline mean ± SD MF values for the L-3/4, L-4/5 and L-5/6 lumbar levels were 218.10 ± 17.81, 200.71 ± 12.63 and 190.10 ± 14.89 Hz, respectively. Following cyclic loading, the MF values decreased from baseline values to 215.40 ± 24.07, 194.75 ± 13.14 and 183.04 ± 14.16 Hz. The MF for L-3/4 and L-5/6 continued to decrease exponentially until 1 h into the recovery period and became significantly lower than baseline 208.43 ± 14.94 and 177.46 ± 16.24 Hz, respectively. The L-4/5 lumbar level MF did not clearly show the exponential decrease in the beginning of the recovery period, as it might have been masked by the large standard deviations. The average percent difference from baseline at the first hour of recovery was 4.4%. Thereafter, the MF increased to baseline values by the 4th hour for L-3/4, the 3rd hour for L-4/5 and after the 2nd hour for L-5/6, after which it continued to exponentially increase above the baseline. In the 5th–7th hours of recovery, MF for the L-4/5 and L-5/6 lumbar levels were significantly higher than baseline being 213.87 ± 14.89, and 209.26 ± 19.64 Hz, respectively (see Fig. 7).

3.5. Models

The models derived for the DNNZ, TNNZ and PMAV are shown superimposed on their corresponding data in Figs. 4–7. The regression coefficient \((r^2)\) for the DNNZ models ranged between 0.9583 and 0.9993, for the TNNZ between 0.9737 and 0.9991, for the PMAV between 0.9337 and 0.9738 and for the MF between 0.9629 and 0.9985.

3.6. Statistics

A square root transformation was applied to the TNNZ and PMAV data for all lumbar levels to obtain a normal distribution. All figures presented in the text were composed using the untransformed data.

4. Discussion

Recalling that increasing NNZ is indicative of decreased stability, the major findings of this investigation consist of the observations of profound changes in the neuromuscular neutral zones (NNZ) and the motor control of muscular activity as a direct response to cyclic loading. In specific, 3 major issues are of importance: cyclic loading elicited a period of 2–3 h post-loading during which there was a significant increase in the NNZ and exposure to instability. During the same 2–3 h post-loading, a significant decrease in the magnitude of muscular activity further compromised the stability of the spine. A new compensatory neural mechanism emerged. This mechanism is capable of allowing muscular activity to trigger significantly earlier and provide more force while the DNNZ are still compromised and not fully recovered together with the laxity in the viscoelastic tissue.

Overall, the first 2–3 h post-cyclic loading are associated with substantial increase in exposure to spinal instability due to laxity in the ligaments together with significant impairment in muscular activity.

The original activation of the multifidus muscles was shown to be a direct reflexive response to stretch of the ligaments and other viscoelastic tissues of the lumbar spine (Stubbs et al., 1998; Solomonow et al., 1998). In essence, elongation or loads above a certain threshold triggered reflexive muscular activity that stiffened the spine. It was further demonstrated that as tension–relaxation or creep develop in the spine over a period of flexion or loading, respectively, the trigger threshold for the reflex shifts substantially (Solomonow et al., 1999). The observed increase in the TNNZ or DNNZ after the cyclic loading period was, therefore, the manifestation of the creep that developed in the viscoelastic tissue. Similarly, the reduction in the PMAV was observed before and is a typical response to development of creep in the tissues during cyclic loading (Solomonow et al., 1998; Hoops et al., 2007; Le et al., 2007).

While the DNNZ approximates the creep recovery over the 7 h rest period, significant differences between these variables exist. The models for the DNNZ recovery predict a time constant in the range of 108.5–276 min while the time constants of the creep recovery were about 50 min (Le et al., 2007). The two- to fivefold larger time-constants for the DNNZ emerge from the fact that the true creep is masked by intervertebral stiffening as a result of the muscular contractions. The measured creep reflects the stiffening whereas the DNNZ reflects the actual displacement where muscular activity was triggered. While one can suggest that the creep is related to the DNNZ, it cannot be relied upon to approximate the displacement NZ, as an artificial fast rate of recovery may be obtained.

It was expected that as the creep in the tissues recovers over the 7 h of rest, the TNNZ, DNNZ, PMAV and the MF will return to normal. The DNNZ indeed returned to near normal level at the 7th

![Fig. 7. The pooled median frequency (MF) mean ± SD for the three lumbar levels before the cyclic loading and during the recovery period. (*) Indicates significant difference from baseline at the respective data point along the time axis.](image)
hour of recovery, yet the behavior of the TNNZ, PMAV and the MF was completely different. Muscular activity was triggered at lower than baseline tension and at a higher level of activation after 2–3 h of rest. The TNNZ continued to decrease below baseline and the PMAV continued to increase above baseline with rest, reaching saturation near the 6th–7th hour. The MF pattern followed that of the PMAV, increasing after the first 2–3 h of recovery and indicating that recruitment of larger motor units is taking place.

The models developed expose the link between the TNNZ, PMAV and MF: activation by the same source. The dominant time constant from mid to end of recovery of the three variables were nearly overlapping; TNNZ, $T_d$ ranged from 100 to 280 min, $T_f$ for the PMAV ranged from 170 to 400 min and $T_{pM}$ of the MF ranged from 171 to 321 min. Essentially, the motor control activation of the compensatory mechanism recruited additional, larger motor units after the 3rd hour of recovery, increasing the average conduction velocity and therefore the MF of the EMG. This was manifested by the corresponding increase of the EMG amplitude, expressed by the PMAV, as well as by the corresponding early increase in the tension, reflected by the TNNZ. Further confirmation that a single compensatory motor control triggered the TNNZ, PMAV and MF could be obtained from the $T_d$ associated with the corresponding models. The $T_d$ values ranged from 224 to 294 min, indicating that the compensatory motor control was initiated and affected all the above parameter simultaneously. Furthermore, the behavior of the TNNZ, PMAV and MF trends could not be explained by a simple ligamento-muscular reflex, as the viscoelastic tissues were still substantially lax by the 2nd or 3rd and up to the 7th hour of recovery. Increasing PMAV and MF with decreasing TNNZ would require tightening of the viscoelastic tissues above their baseline to elicit such an increase as a reflexive response. Based on the observations above, it is becoming apparent that a different, compensatory neural control mode is activated 2–3 h after cyclic loading.

The different neural control modes could be associated with the clinical finding that tissue damage and the associated pain results in spasms, elevated muscular activity and joint stiffness (Pedersen et al., 1956; van Dien et al., 2003). The work of Woo et al. (1981, 1982) demonstrated that cyclic creep of viscoelastic tissues within the physiologic range is associated with microdamage in the collagen fibrils. The viscoelastic tissue could be considered damaged in this experiment as it was exposed to 60 min of cumulative cyclic loading. This neural control mode, therefore, may be triggered by the tissue damage and the associated pain mechanism.

The micro-damage in the viscoelastic tissues could be classified as sub-clinical for such physiological loads and displacements, yet was shown in humans to result in stiffening of the spine for several hours after the work was completed (Granata and Marras, 2000). Similarly, Olson et al. (2004, 2006), Li et al. (2007), Shin and Mirka (2007), Granata et al. (2005), Dickey et al. (2003) found that such significant changes in muscular activity occur after moderate and mild cyclic loading in humans. In essence, these complementary findings validate that the observations made in this project, using an in vivo cat model, are also seen in humans subjected to similar loading conditions, and that mild loading dose duration can trigger such muscular responses.

In conclusion, a period of cyclic loading significantly increased the tension and displacement NZ while decreasing peak muscular activity in the 2–3 h immediately after the work. This suggests that the lumbar spine is exposed to significant reduction in stability control in that period. A compensatory neural control mechanism is triggered by the 3rd hour post-cycling loading, and significantly enhanced the magnitude and timing of the muscular contributions while allowing the viscoelastic tissues to recover from the imposed creep for several hours longer.

Conflict of interest statement

There is no conflict of interest regarding the publication of this paper.

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