Motor control of lumbar instability following exposure to various cyclic load magnitudes

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Abstract The motor control system may compensate for lumbar instability following cyclic work with differential response to load magnitude. In vivo felines were exposed to a cumulative 1 h of cyclic work at 0.25 Hz. One group exposed to light whereas the second to heavy load while recording lumbar displacement and multifidus EMG during work and in single test cycles over 7 h rest post-work. Significant laxity and reduced reflexive EMG activity were evident immediately post-work in both groups. EMG and laxity recovered over 7 h rest in the group exposed to light load whereas in the group exposed to heavy load, motor control compensation was triggered within 1–2 h post-work. The compensation was expressed by earlier and stronger muscular activation than in baseline. It is concluded that cyclic work is deleterious to spine stability immediately after work. Work with heavy loads elicits delayed motor control compensation whereas work with light loads leaves the spine unstable and exposed to injury for several hours. Overall, prolonged cyclic or repetitive work elicits a transient instability disorder, regardless of the load handled, exposing the individual to potential injury.

Keywords Spine · Lumbar · Stability · Muscles · EMG · Multifidus · Ergonomics · Disorder

Introduction

The epidemiology identifies high rates of cumulative trauma disorder (CTD) in workers engaged in repetitive/cyclic occupational activities (loading/unloading boxes, assembly line, construction, etc.) [10, 26, 39, 40]. Recent research utilizing an in vivo model confirmed the epidemiology and provided the biomechanical and neurophysiological etiology of the lumbar disorder. High loads [17], many repetitions [30], short rest durations [11] and fast rates of flexion [22, 23] were shown to induce substantial creep in the lumbar viscoelastic tissues (ligaments, discs, capsules and dorsolumbar fascia). The associated micro-damage in the collagen fibers [57–59] triggered an acute inflammation [19], which in turn triggered a neuromuscular disorder [50]. Additional research with human subjects performing cyclic lumbar flexion confirmed the acute neuromuscular disorder and creep observed in the in vivo models [3, 6–8, 16, 20, 21, 28, 31–33, 42]. It is expected that the substantial creep and neuromuscular disorder observed in the above studies may also be associated with large changes in lumbar stability, an issue which is yet to be investigated.

Stability of the lumbar spine is a complex mechanism consisting of passive (ligaments, disks, capsules and fascia) and active (musculature) components, multi-feedback loops and motor control [1, 24, 27, 35–37, 41]. Panjabi [35] designated the small passive perturbations of the intervertebral joint about the neutral position within which the viscoelastic tissues were not engaged/active as the neutral zone (NZ). Similarly, the small increases in passive displacement or tension to the intervertebral joint which did not trigger reflexive muscular activity were designated as the neuromuscular neutral zones (NNZ) [4, 18, 49, 51, 60]. Therefore, increases in the displacement or tension NNZ

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above normal are indicative of decreasing stability. Under such conditions, the vertebrae move to a larger extent without muscular protection. Conversely, decreasing displacement or tension NNZ below normal are considered as increasing lumbar stability, as the musculature becomes active in response to smaller movements or smaller loads than normal.

The objective of this study is to identify the changes in lumbar stability following the exposure to prolonged cyclic loads of different magnitudes. We hypothesize that cyclic load of low magnitude will elicit increases in the NNZs (or increase instability) and will gradually recover to normal over several hours of rest whereas exposure to cyclic load of high magnitude will elicit significant increase in the NNZs (or significantly increased instability) that will require motor control compensation with early and powerful activation of the musculature. It is anticipated that such information will provide new insights on the motor control mechanism of lumbar stability, the exposure to instability and potential injury following cyclic activities and the basis for design of safe work programs and prevention of injury.

Methods

Preparation

Seventeen adult cats, with an average weight of 4.03 ± 1.1 kg, were used in this study. The cats were separated into two experimental groups (see below in *Protocol*). They were anesthetized with 60 mg/kg chloralose, according to a protocol approved by the Institutional Animal Care and Use Committee. Animals remained anesthetized throughout the experiment (approximately 9 h). A superficial skin incision overlying the lumbar spine was made to expose the dorsolumbar fascia, and an S-shaped stainless steel hook made of 1.5-mm-diameter rod was applied around the supraspinous ligament between L4 and L 5. The preparation was then positioned in a rigid stainless steel frame and the lumbar spine was isolated by means of two external fixators, which were applied to the L1 and L7 posterior processes, respectively. The external fixation was intended to limit the elicited flexion to the lumbar spine and to prevent interaction of thoracic and sacral and/or pelvic structures, but not to prevent any motion. Fixators remained in place throughout the experiment (approximately 9 h).

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 1,000 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 1,000 Hz, along with the EMG signals.

Under such loading condition, the lumbar spine underwent anterior flexion-extension while straining the supraspinous, intraspinal, and posterior longitudinal ligaments as well as the ligamentum flavum, facet capsule and dorsolumbar 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. Schematic diagram and X-ray verification of the elicited flexion-extension are available in a previous report [56]. Overall, the neuromuscular response represents the reflexive activation from afferents in several lumbar viscoelastic components similar to those active during flexion-extension.

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. A set of six 10-min cyclic loading periods at 0.25 Hz and 20 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 20 N peak load at 0.25 Hz were applied. The single cycles were applied at 10, 30 and 60 min following termination of the cyclic loading period and then once every hour. Overall, nine test cycles were applied during the 7 h recovery period. Figure 1 (bottom trace) shows the schematic of the cyclic loading. EMG, load and displacement were recorded throughout the protocol.

The average of the first two cycles during the first 10 min of loading in each animal was used as the baseline to establish the normal neuromuscular neutral zones (NNZ) before prolonged cyclic loading.

The same loading protocol was repeated in a different group of animals with the peak load of 60 N. Based on

Fig. 1 Typical recording of the cyclic load (*bottom*), displacement (above the bottom trace), EMG of the three lumbar levels (*top three traces*) and the test cycles collected over the 7 h recovery period. Note: the bottom plot is also a display of the loading protocol

previous studies, 20 N was found to be a light load whereas 60 N was considered as a high load, near maximal [46]. For the 20 N load group, $N = 8$ and for the 60 N group, $N = 9$.

Data analysis

The analysis considered the recorded EMG, vertical displacement and cyclic load applied to the spine during the first two cycles of the first 10 min of loading and the nine individual test 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 EMG signal level at rest. The mean absolute value (MAV) of this resting level was calculated: the signal recorded from each lumbar level was full wave rectified and 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 resting 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) threshold values for that particular trial and lumbar level. Similarly, as the EMG dropped below the three times resting MAV threshold in the relaxation 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 spasms along the baseline, or the EMG discharge during the flexion-extension was below three times resting level, 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 ($\pm$ SD) were calculated and plotted as a function of time. The DNNZ and TNNZ of the first two cycles of the first 10 min of loading were averaged for each preparation to yield a mean and SD, and then pooled with those of the other preparations to use as the NNZ baselines.

The mean peak MAV (PMAV) of the first two cycles was used as a normalization value for the peak MAV recorded in the test cycles of the same preparation during the 7 h recovery period. A single peak value was determined for each cycle. The normalized values of all the preparations were pooled, and the mean ($\pm$ SD) was calculated and plotted as normalized peak MAV versus time to assess whether the contraction level increased or decreased because of cyclic loading.

EMG median frequency (MF) was found for each single-cycle test in order to identify changes in motor unit recruitment [45]. A 500 ms window, centered at the peak load of each cycle, within which EMG was approximated as a stationary signal, and was zero-padded on both sides. A Tukey window was applied to the zero-padded data, the power spectral density of the signal in this window was found via the fast Fourier transform, and the MF, defined as the frequency that divides the area under the power spectral density in half, was calculated. The MF values of all corresponding time points and lumbar levels were pooled and plotted as mean MF versus recovery time with the mean MF of the first two cycles as baseline.

Statistics

Since multifidus segments are not innervated from multiple roots [25], the data were divided into three lumbar levels (L3–4, L4–5 and L5–6) for statistical analyses. The DNNZ, TNNZ, and peak MAV 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 (baseline, 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 (baseline, recovery times) and the dependent variables were normalized peak MAV and median frequency. Upon determining a significant interaction or main effect, pair wise comparisons were performed using a Student's *t* test. The level of significance for all tests was set as $p = 0.05$.

Modeling

The mean $\pm$ SD values of the DNNZ, TNNZ, peak MAV and MF during recovery for each lumbar level were fit with exponential-based models, as they represent the classical response of viscoelastic tissues [48].

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

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

where D_0 is the intercept of the displacement (mm), D_R is the residual displacement, relative to baseline, at the end of recovery (mm), D_L is the displacement, relative to baseline, at the beginning of recovery (mm), τ_1 is the time constant

of the exponential decay (min), τ_r is the time of the first recovery measurement (120 min).

As noted above, the model was evaluated from the time point of 120–530 min, which constitutes the 7 h of recovery following the cyclic loading period.

The time-course of the TNNZ thresholds during the stretch and relaxation phases of the test cycles during the recovery period were described by

$$\text{TEN}(t) = T_0 + (t - \tau_r)T_L \left(e^{-\frac{t-\tau_r}{\tau_2}} \right) + T_M \left(e^{-\frac{t-\tau_r}{\tau_3}} \right) \quad \{t|120 \leq t \leq 530\} \quad (2)$$

where T_0 is the intercept of the tension (N), T_L affects the rise amplitude (N/s), T_M is the amplitude of the decay dominating the end of the recovery period (N), $(t - \tau_r)T_L \left(e^{-\frac{t-\tau_r}{\tau_2}} \right)$ allows for a transient rise at the beginning of the recovery period, τ_2 affects the rates of rise and fall (s), 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 was described by

$$\begin{aligned} \text{peakMAV}(t) &= \begin{cases} P_0 + P_L \left(e^{-\frac{t-\tau_r}{\tau_4}} \right) + P_M \left(1 - e^{-\frac{t-\tau_r}{\tau_5}} \right), & t < \tau_d \\ P_0 + P_L \left(e^{-\frac{t-\tau_r}{\tau_4}} \right) + P_M \left(1 - e^{-\frac{t-\tau_r}{\tau_5}} \right) + (t - \tau_d)P_H \left(e^{-\frac{t-\tau_d}{\tau_6}} \right), & t \geq \tau_d \end{cases} \quad \{t|120 \leq t \leq 530\} \end{aligned} \quad (3)$$

where P_0 is the intercept of the peak MAV, P_L is the amplitude of the exponential decay, P_M is the amplitude of the exponential increase, τ_4 , τ_5 are exponential time-constants of the terms dominating the beginning of recovery (min), τ_6 is the exponential time-constant of the hyperexcitability term dominating end of recovery (min), $(t - \tau_d)P_H e^{-\frac{t-\tau_d}{\tau_6}}$ is the hyperexcitability term. This term has a delayed onset during the recovery period and is equal to zero when $t < \tau_d$, τ_r is the time of the first recovery measurement (120 min).

The time-course of the MF data during the recovery was described by

$$\text{MF}(t) = \begin{cases} F_0 + F_L \left(e^{-\frac{t-\tau_r}{\tau_7}} \right) + F_M \left(1 - e^{-\frac{t-\tau_r}{\tau_8}} \right), & t < \tau_d \\ F_0 + F_L \left(e^{-\frac{t-\tau_r}{\tau_7}} \right) + F_M \left(1 - e^{-\frac{t-\tau_r}{\tau_8}} \right) + (t - \tau_d)F_H \left(e^{-\frac{t-\tau_d}{\tau_9}} \right), & t \geq \tau_d \end{cases} \quad \{t|120 \leq t \leq 530\} \quad (4)$$

where F_0 is the intercept of the peak MF (Hz), F_L is the amplitude of exponential decay seen at the beginning of the

recovery period (Hz), F_M is the amplitude of exponential increase following the decay in the recovery period (Hz), τ_d is the time of onset of hyperexcitability/compensation (min), τ_7 , τ_8 are exponential time-constants of the terms dominating the beginning of recovery (min), τ_9 is the exponential time-constant of the hyperexcitability term dominating end of recovery (min), $(t - \tau_d)F_M e^{-\frac{t-\tau_d}{\tau_{10}}}$ is the hyperexcitability term with delayed onset dominating end of recovery period (equal to zero when $t < \tau_d$), τ_r is the time of first recovery measurement (120 min).

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 specimen undergoing 60 N cyclic loading is shown in Fig. 1. Figure 2 displays the load, displacement and EMG of the multifidi of a single level delineating the definitions of the displacement NNZ (DNNZ) and tension NNZ (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 $\pm$ SD of the DNNZ for each loading condition (20 N, 60 N) before and after cyclic loading.

For the 20 N loading condition, each of the lumbar levels demonstrated an increase immediately following the

cyclic loading period and then gradually decreased toward baseline values. Statistical analyses revealed no phase $\times$ time interactions for the L3–4 and L4–5 levels ($p = 0.360$, $p = 0.769$) along with significant main effects of time ($p < 0.001$ for each level) and a significant interaction for the L5–6 level ($p = 0.048$). The interaction in L5–6 occurred because the DNNZs during the stretch phase did not return to baseline while the relaxation phase did during the last hour of the recovery period (Fig. 3). Baseline values for the stretch phase were 2.7, 2.0, and 2.1 mm for the L3–4, L4–5 and L5–6 levels, respectively. These values had increased approximately 2.4–3.3 times when measured immediately after the cyclic loading. Baseline relaxation values were 4.3, 4.7 and 4.6 mm, respectively, and increased approximately 2.0–2.2 times when measured after the cyclic loading. During the recovery period DNNZs decreased to 3.5, 3.3 and 3.6 mm (stretch phase), respectively, and 5.9, 5.7 and 5.4 mm (relaxation phase), respectively, by the end of the recovery period.

The 60 N loading condition also demonstrated an increase measured immediately after the cyclic loading period followed by a gradual decrease toward baseline values. There were no phase $\times$ time interactions for any of the lumbar levels ($p = 0.605$, $p = 0.632$, $p = 0.619$), and all levels demonstrated a significant main effect of time ($p < 0.001$ for all levels). The mean baseline values for the stretch phase were 6.1, 6.0, and 5.6 mm for the L3–4, L4–5, and L5–6 levels. These values had increased approximately 2.2–2.3 times when measured immediately after the cyclic loading period. The mean baseline relaxation values were 9.9, 9.7 and 9.8 mm, respectively, and increased approximately 1.7–1.8 times above baseline when measured after cyclic loading. The DNNZs remained significantly elevated more than 4 h following cyclic loading at the L3–4 and L4–5 lumbar levels, and more than 5 h at the L5–6 lumbar level. By the end of the recovery period the mean DNNZs had decreased to 5.9, 6.1, and 5.3 mm (stretch phase) and 11.1, 11.2, and 12.2 mm (relaxation phase) and were not significantly different from the baseline.

Tension neuromuscular neutral zones (TNNZ)

Figure 4 displays the mean $\pm$ SD of the TNNZ for each loading condition (20 and 60 N) before and after cyclic loading.

In the 20 N loading condition, there were no phase $\times$ time interactions for any of the L3–4 and L4–5 lumbar levels ($p = 0.390$, $p = 0.881$). These levels demonstrated significant main effects of phase ($p < 0.001$ for each level) and time ($p < 0.001$ for each level). A significant phase $\times$ time interaction ($p = 0.030$) was found in the L5–6 level since the TNNZ for the stretch phase did not

Fig. 2 Typical recording of load, displacement and EMG collected during a 60N test cycle. Graphical definitions of the DNNZ, TNNZ and PMAV are superimposed on the collected data

Fig. 3 The mean $\pm$ SD displacement neuromuscular neutral zones (DNNZ) of the baseline and the test cycles during the 7 h rest following cyclic loading is shown for the three lumbar levels. The model developed is superimposed on the data points. Significant differences from baseline values are highlighted with asterisks. The r^2 corresponding to the model is in the inset

Fig. 4 The mean $\pm$ SD tension neuromuscular neutral zones (TNNZ) of the baseline and of the test cycles during the 7 h rest following cyclic loading is shown for the three lumbar levels. The model developed is superimposed on the data points. Significant differences from baseline values are highlighted with asterisks. Note that statistical significance is present in L5-6 for the first 2 h of recovery in the relaxation phase of the 20 N set. The r^2 corresponding to the model is in the inset

change significantly following the cyclic loading period while the TNNZs measured during the relaxation phase demonstrated a similar significant change pattern as the L3–4 and L4–5 levels. The baseline values for the stretch phase were 5.0, 3.8 and 4.1 N for the L3–4, L4–5 and L5–6 levels, respectively. These values increased approximately 1.6 and 1.9 times above baseline within 30 min following the cyclic loading period in the L3–4 and L4–5 lumbar levels only. The baseline relaxation values were 9.7, 8.7 and 7.9 N, respectively, and increased approximately 1.4–1.5 times above baseline within 30 min following the cyclic loading period. The TNNZs remained significantly elevated through 1, 2, and 2 h during the recovery period for each of the lumbar levels, respectively. Afterward the TNNZ values decreased below baseline, although not statistically significant to 3.9, 3.8, and 4.7 N (stretch phase) and 7.8, 6.8, and 5.5 N (relaxation phase) by the end of the recovery period.

There were no phase $\times$ time interactions in the 60 N loading condition for any of the lumbar levels ($p = 0.792$, $p = 0.889$, $p = 0.791$), but demonstrated significant main effects of time ($p < 0.001$ for all levels). The baseline values for the stretch phase were 21.3, 20.3 and 18.9 N for L3–4, L4–5 and L5–6 levels, respectively. The baseline values for the relaxation phase were 37.2, 33.3 and 35.7 N for L3–4, L4–5 and L5–6 levels, respectively. Each lumbar

level demonstrated significant increases of 1.5, 1.4, and 1.3 times (stretch) and 1.2, 1.2, and 1.3 times (relaxation) above baseline values within 10 min following the cyclic loading period. The TNNZs remained significantly elevated for 1 h in L3–4 and for 2 h in L5–6, but decreased back to baseline by the end of the first hour in L4–5. The TNNZs for each lumbar level significantly decreased below baseline by the fifth hour of the recovery period to final values of 7.6, 9.2 and 5.2 N (stretch phase) and 20.5, 22.8 and 28.8 N (relaxation phase) for L3–4, L4–5 and L5–6 levels, respectively.

For each lumbar level, both the DNNZs and TNNZs measured during the stretch phase of loading were significantly smaller than those measured during the relaxation phase of loading ($p < 0.001$ for all levels). The differences between the loading phases have been discussed in detail in a previous publication [49].

EMG peak mean absolute value (PMAV)

Figure 5 displays the mean $\pm$ SD of the PMAV for each loading condition (20 and 60 N), before and after the cyclic loading period.

In the 20 N loading condition, significant changes with time were found in L3–4 ($p = 0.004$) and L4–5 ($p = 0.040$) levels while L5–6 did not vary significantly

Fig. 5 The mean $\pm$ SD Peak MAV (PMAV) of the baseline and of the test points during the 7 h rest following the cyclic loading is shown for the three lumbar levels. The model developed is superimposed on the data points. Significant differences from baseline values are highlighted with asterisks. The r^2 corresponding to the model is in the inset

with time ($p = 0.114$). Pair wise comparisons revealed a significant decrease to 68% and 69% of the baseline values within the first hour of recovery for the L3–4 and L4–5 levels, respectively. Following this decrease, there was a gradual, but insignificant, increase in the mean values to baseline and further above, although not statistically significant.

In the 60 N loading condition, significant decreases to below baseline were found at each lumbar level ($p < 0.001$ for all levels) when measured immediately after the cyclic loading period. These values decreased to 64, 66, and 66% of baseline within the first hour and returned to baseline before the third hour of the recovery period. A further increase above baseline by the third hour of the recovery period was also present, although not statistically significant.

EMG median frequency (MF)

Figure 6 displays the mean $\pm$ SD of the MF for each loading condition (20 and 60 N) before and after cyclic work.

In the 20 N loading condition, significant changes with time were found at the L4–5 level ($p = 0.008$) while no changes were found with time at the L3–4 ($p = 0.268$) and L5–6 ($p = 0.150$) levels. Pair wise comparisons revealed a

10% decrease in MF at the L4–5 level measured after the cyclic loading period and a gradual increase to baseline by the fourth hour of the recovery period.

For the 60 N load, L4–5 and L5–6 demonstrated significant changes with time ($p = 0.025$ and $p < 0.001$) while L3–4 did not change ($p = 0.123$). The MF significantly decreased in L5–6 within 30 min following cyclic work and gradually increased to the baseline after the first hour of the recovery period. Both L4–5 and L5–6 demonstrated a significant increase in MF at the sixth and seventh hours during the recovery period.

Models

Empirical models optimized to fit the DNNZ and TNNZ data are superimposed on Figs. 3, 4, 5 and 6. The regression coefficient (r^2) ranged from 0.970 to 0.992 for the DNNZ and from 0.791 to 0.987 for the TNNZ in the 20 N loading condition. The regression coefficient ranged from 0.953 to 0.997 for the DNNZ and from 0.918 to 0.989 for the TNNZ in the 60 N loading condition. The regression coefficient for the PMAV ranged from 0.412 to 0.761 and 0.407 to 0.998 for the 20 and 60 N loading conditions, respectively. The regression coefficient for the MF ranged from 0.805 to 0.911 and 0.991 to 0.993 for the 20 and 60 N loading conditions, respectively.

Fig. 6 The mean $\pm$ SD median frequency (MF) of the baseline and of the test points during the 7 h rest period following the cyclic loading is shown for the L3–4, L4–5 and L5–6. The model developed is superimposed on the data points. Significant differences from baseline values are highlighted with asterisks. The r^2 corresponding to the model is in the inset

Table 1 Optimized parameters for the empirical models describing behavior of the displacement neuromuscular neutral zones (DNNZ) versus time during the experiment $DISP(t) = D_0 + D_R + (D_L - D_R)e^{[-(t-\tau_r)/\tau_1]}$

Load	Phase	D_0	D_R	D_L	τ_1
L ₃₋₄ lumbar level					
20 N	Stretch	2.76	0.79	3.79	129
	Relaxation	2.39	0.11	7.21	518
60 N	Stretch	4.59	-0.79	9.03	255
	Relaxation	7.54	-3.11	9.97	666
L ₄₋₅ lumbar level					
20 N	Stretch	1.84	1.17	4.52	206
	Relaxation	3.28	0.09	6.52	411
60 N	Stretch	5.61	-0.24	7.02	154
	Relaxation	7.35	-2.33	9.83	600
L ₅₋₆ lumbar level					
20 N	Stretch	2.10	1.39	3.67	122
	Relaxation	3.30	-0.57	6.28	445
60 N	Stretch	4.13	-1.48	7.45	321
	Relaxation	7.50	-0.95	9.83	643

Table 2 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						
20 N	Stretch	3.97	0.16	3.25	22	203
	Relaxation	4.75	0.10	7.60	131	131
60 N	Stretch	-2.45	0.00	34.44	-	312
	Relaxation	13.64	0.45	30.2	115	114
L ₄₋₅ lumbar level						
20 N	Stretch	0.15	0.02	7.09	35	657
	Relaxation	2.43	0.04	10.34	86	500
60 N	Stretch	5.81	0.00	20.76	-	215
	Relaxation	16.93	0.48	24.24	111	58
L ₅₋₆ lumbar level						
20 N	Stretch	4.46	0.08	0.52	34	50
	Relaxation	-0.61	0.06	10.98	130	500
60 N	Stretch	-16.98	0.00	39.53	-	677
	Relaxation	26.85	0.68	19.61	76	31

Optimized parameters for the empirical models are shown in Tables 1, 2, 3 and 4.

Statistics

A log transformation was applied to PMAV data from both loading conditions to obtain a normal distribution. All figures presented in the display untransformed data.

Discussion

The highlights of the findings obtained in this investigation consist of the following.

Regardless of the load level, the first 1–5 h post-cyclic work finds the lumbar spine with substantially decreased stability due to significant viscoelastic tissues laxity simultaneously with significantly attenuated muscular activity. Motor control compensation triggers muscular activity that is higher than normal and occurs earlier in the stretch phase and remains longer in the relaxation phase lending high stiffness to the spine to offset the laxity. The motor control compensation is directly related to the load level, triggering earlier and staying longer as the cyclic load magnitude increases. Finally, cyclic work under light loads does not benefit from the neuromuscular compensation, leaving the spine exposed to instability for several hours.

The DNNZ associated with the low load group exhibited over twofold increase immediately after the cyclic work for both stretch and relaxation phases. The exponential decrease was slow and remained significantly above baseline throughout the 7 h of rest post-work. The TNNZ immediately after cyclic work was nearly 50% larger than baseline for the stretch and relaxation phase. The exponential decrease of the TNNZ reached near the baseline 3–7 h post-work in the three lumbar levels for the stretch and relaxation phases, without decreasing significantly below baseline. The MF indicated that a significant decrease in the active motor units (only in L4–5) was present during the first 3 h immediately after work and gradually increased to asymptotically reach baseline near the end of the 7-h rest. Similarly, the PMAV experienced a significant (L3–4 and L4–5) decrease post-cyclic work and gradually, but not significantly, recovered to just above baseline near the end of the seventh hour. Motor control compensation was absent, as no statistically significant increases in the PMAV, MF or significant decreases of the TNNZ above and below the baseline, respectively, were evident. Overall, despite the light load applied to this group, a substantial laxity and attenuated muscular activity were present in the lumbar spine for several hours following cyclic work, exposing the spine to potential instability.

For the group subjected to the heavy load, the DNNZ increased significantly and substantially, about 1.5–2.3-fold above baseline, during the 3–4 h immediately after the cyclic work for the stretch and relaxation phases. The DNNZ in the last 2–3 h of the 7 h recovery period, post-cyclic work, was near or just below the baseline for the stretch phase and just above the baseline for the relaxation phase. The TNNZ also showed significant and substantial increase (about 1.2–1.5-fold above baseline) during the 1–2 h immediately after cyclic work, for the stretch and

Table 3 Optimized parameters for the empirical models describing behavior of the peak mean absolute value (PMAV) versus time during the recovery period $\text{peakMAV}(t) = P_0 + P_L e^{[-(t-\tau_L)/\tau_4]} + P_M(1 - e^{[-(t-\tau_L)/\tau_5]}) + (t - \tau_d)P_H e^{[-(t-\tau_d)/\tau_6]}$

Load	P_0	P_L	P_M	τ_4	τ_5	P_H	τ_6	τ_d
L _{3–4} lumbar level								
20 N	−3.53	4.20	5.80	152	241	−0.002	600	364
60 N	−2.85	3.59	4.06	62	79	0.001	600	319
L _{4–5} lumbar level								
20 N	−5.28	6.00	6.33	67	74	0.000	600	295
60 N	−6.01	6.84	7.14	40	46	0.001	600	253
L _{5–6} lumbar level								
20 N	−4.50	5.27	6.11	156	189	−0.001	600	372
60 N	−2.79	3.62	4.09	58	80	0.004	394	288

Table 4 Optimized parameters for the empirical models describing behavior of the peak median frequency (MF) versus time during the recovery period $\text{MF}(t) = F_0 + F_L e^{[-(t-\tau_L)/\tau_7]} + F_M(1 - e^{[-(t-\tau_L)/\tau_8]}) + (t - \tau_d)F_H e^{[-(t-\tau_d)/\tau_9]}$

Load	F_0	F_L	F_M	τ_7	τ_8	F_H	τ_9	τ_d
L _{3–4} lumbar level								
20 N	00.0	220.6	227.5	100	108	0.061	160	325
60 N	117.1	90.9	101.5	40	47	0.003	590	140
L _{4–5} lumbar level								
20 N	−297.2	496.1	511.4	97	100	0.004	300	350
60 N	20.5	170.7	175.2	17	18	0.107	500	272
L _{5–6} lumbar level								
20 N	−87.1	272.1	284.8	39	39	0.003	300	350
60 N	22.0	163.2	182.0	127	146	0.060	189	289

relaxation phases. Past the second hour following the cyclic work, the TNNZ decreased below baseline, and after the fifth hour of the 7-h recovery period, reached significance at values corresponding to 50–75% decrease. The PMAV and MF confirm an initial significant muscular activity decrease and motor unit de-recruitment, respectively. These were followed by an increase in muscular activity, re-recruitment of de-activated motor units and recruitment of new, larger motor units near the last 3 h of the recovery period. Overall, motor control compensation seemed to be initiated almost 1–2 h post-cyclic work and substantially increased the muscular activity over the last 5 h. The increase in the muscular activity had two components; earlier activation of the muscular activity during the stretch phase with longer presence of activity in the relaxation phase (as reflected from the decreasing TNNZ), as well as stronger and more powerful contraction (relative to baseline) as evident from the higher PMAV and MF. The increased muscular activity seems to compensate for the laxity of the viscoelastic tissues by increasing the stiffness of the intervertebral joints. Indeed, the faster decrease of the DNNZ of the group subjected to heavy

loads and its arrival to just below baseline levels was probably due to the increased muscular activity, lending higher stiffness to the spine whose viscoelastic tissues were still lax. It should be noted that despite the significant effects of the motor control compensation on lumbar stiffness, the first 1–2 h immediately post-cyclic work left the spine with substantial decrease in stability and exposed to injury.

Examination of the time constants associated with the models derived for the data confirms that the changes in the TNNZ, PMAV and MF occurred at about the same time. The time delay, τ_d , for the PMAV ranged from 252 to 318 min, and for the MF it ranged from 272 to 289 min in the L3–4 to L5–6, respectively. It is evident that the triggering time of the compensatory motor control was reflected from all parameters, where re-recruitment of the de-activated motor units followed by additional recruitment of new, larger units (MF) brought about a substantial increase in muscular activation level (MAV) applied earlier and remaining longer during the flexion-extension cycle, respectively, decreasing the TNNZ and stiffening the spine (DNNZ).

In a previous, preliminary report [51], we identified the motor control compensation for lumbar instability following cyclic work at a moderate load of 40 N. The TNNZ demonstrated a decrease to below baseline after the second to third hour post-cyclic work as compared to the first to second hour for the extreme load of 60 N seen in the present investigation. Similarly, the PMAV and MF in the 40 N load exceeded the baseline past the second to third hour post-work. Combining the previous data with the new data presented here confirms that a direct relationship may exist between the load magnitude handled during the cyclic work and the time at which the motor control compensation triggers. Heavy loads trigger earlier muscular compensation, about 1–2 h post-work, whereas, moderate loads trigger the same effect within 2–3 h post-work. Light loads, however, demonstrate a very mild and statistically insignificant compensatory activity, leaving the spine exposed to instability for over 5 h.

The source of the neuromuscular compensation is of interest. As the viscoelastic tissues are lax due to the creep developed in the course of the cyclic work, the ligamentomuscular [52], as well as facet capsule-muscular [2, 14, 15] reflexes are severely attenuated and non-functional [47]. On the other hand, the viscoelastic tissues were subjected to a certain level of micro-damage [57–59] and probably developed an acute inflammation [19, 50] which in turn may trigger the pain receptors in these tissues to initiate a protective muscular activity. Indeed, the presence of high level EMG and its associated lumbar stiffness in individuals with confirmed low back injuries/pain is well established [5, 9, 12, 29, 43, 53]. Most likely, the presence of some level of tissue damage is the triggering source of the neuromuscular compensation as it seems to be present in conditions that were shown before to be inductive of inflammatory responses [11, 17, 22, 23, 30].

The data presented above identify the need for the definition of a new disorder; transient instability disorder (TISD). The TISD is defined as a transient significant decrease in the contribution of the viscoelastic tissues concurrently with a decrease in the contribution of the musculature, resulting in lumbar laxity that may expose the intervertebrae joints to instability. The epidemiological evidence [10, 26, 39, 40] as well as the biomechanical and neurophysiological confirmation [11, 17, 22, 23, 30] asserts that cyclic or repetitive work while handling light loads, does not lead to a cumulative trauma disorder (CTD) over time. Nevertheless, prolonged, repetitive motion under light loads is shown here to trigger a transient lumbar instability that can expose the spine to injury as a result of even routine movements. It is essential that the reader realizes that CTD and transient instability disorder (TISD) are two distinct disorders. The first is a chronic disorder leading to viscoelastic tissue degeneration over time and

the loss in the associated function [19], whereas the TISD is a transient disorder, where the stability of the spine is compromised for several hours' post-cyclic work. Overall, cyclic/repetitive work under low/light loads is not a "risk free" or "non-risk" activity. It may not lead to CTD, but will render the spine to a transient instability period.

Experimental research using animal models allows performance of invasive procedures that cannot be applied to humans and can also allow isolation of stimulus and responses not readily obtained from humans. While such animal research is insightful and produces distinct progress in medical knowledge, it needs to be confirmed as applicable to humans. In this case, cats are well established as a valid model in neuromuscular physiology relative to reflexes, motor control, neuromuscular responses etc. [38]. Biomechanically, the quadruped was also shown to be a valid model in spine research. Ianuzzi et al. [13] established scaling for size differences in cats versus humans for intervertebral angles, joint movements, yield points and torque limits, closing the gap for size differences. Williams et al., have [56] also shown that the physiological strain of cat supraspinous ligaments is nearly identical to that of humans [34] requiring no scaling conversion as it is expressed in percent elongation. Smit [44] and Wilke et al. [54, 55] also validated the use of quadruped spine as an appropriate model for a human spine. Recently, the quadruped spine was also validated for vertebral osteoporosis [61]. Overall, neurophysiologically and biomechanically the quadruped spine is established as a valid model for spinal research with a size scaling for some mechanical data. One should expect that data obtained from validated animal models will apply to humans in the principal of behavior/response and scaling for size.

Conclusions

Based on the data presented in this report, the following conclusions can be made for the feline under the described conditions:

1. Regardless of the load magnitude, prolonged cyclic work leaves the lumbar spine transiently unprotected due to laxity of the passive tissues concurrently with low activation of the musculature. The two mechanisms which maintain lumbar stability are temporarily deficient, exposing the spine to potential injury.
2. Cyclic work at high load benefits from motor control compensation within 1–2 h after work was terminated. The compensation is expressed by earlier and more powerful activation of the muscles during the flexion phase and longer presence during the extension phase. The compensation lends the necessary stiffness to the

spine to offset the lost contributions of the viscoelastic tissues which require over 7 h for recovering to near normal function.

3. The motor control compensation seems to be directly related to the load magnitude handled during the cyclic work. High load magnitude benefits from early activation of the compensation whereas reduced load triggers the compensation later.
4. Cyclic work at low load triggers laxity in the viscoelastic tissues and deficient muscular activation that lasts for several hours without benefiting from motor control compensation.

Overall, the immediate 1–3 h after completion of repetitive work requires protection from instability. While lifting low loads in a cyclic/repetitive mode was not identified by the epidemiology nor by biomechanical and neurophysiological data to be a risk factor for cumulative trauma disorder, the data obtained in this report, however, issues a clear warning that cyclic work at low loads can render the spine unprotected from instability for many hours after work.

Important support for the possible presence of such instability post-cyclic physical work in humans comes from clinical experience. Patients often complain that they performed 8 h of physical work without a problem, yet, at the end of the day, upon changing clothes, they bent over to put on shoes and something “clicked” in their back, leaving them with pain and disability. Such common and familiar complaint from humans receives a very sound explanation from our data, although obtained from the felines under laboratory loading.

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