SPINE Volume 31, Number 7, pp 839–845

Disturbed Paraspinal Reflex Following Prolonged Flexion-Relaxation and Recovery

Ellen L. Rogers, and Kevin P. Granata, PhD

Study Design. Repeated measures experimental study of the effect of flexion-relaxation, recovery, and gender on paraspinal reflex dynamics.

Objective. To determine the effect of prolonged flexion-relaxation and recovery time on reflex behavior in human subjects.

Summary of Background Data. Prolonged spinal flexion has been shown to disturb the paraspinal reflex activity in both animals and human beings. Laxity in passive tissues of the spine from flexion strain may contribute to desensitization of mechanoreceptors. Animal studies indicate that recovery of reflexes may take up to several hours. Little is known about human paraspinal reflex behavior following flexion tasks or the recovery of reflex behavior following the flexion tasks.

Methods. A total of 25 subjects performed static flexion-relaxation tasks. Paraspinal muscle reflexes were recorded before and immediately after flexion-relaxation and after a recovery period. Reflexes were quantified from systems identification analyses of electromyographic response in relation to pseudorandom force disturbances applied to the trunk.

Results. Trunk angle measured during flexion-relaxation postures was significantly higher following static flexion-relaxation tasks ($P < 0.001$), indicating creep deformation of passive supporting structures in the trunk. Reflex response was diminished following flexion-relaxation ($P < 0.029$) and failed to recover to baseline levels during 16 minutes of recovery.

Conclusion. Reduced reflex may indicate that the spine is less stable following prolonged flexion-relaxation and, therefore, susceptible to injury. The absence of recovery in reflex after a substantial time indicates that increased low back pain risk from flexion-relaxation may persist after the end of the flexion task.

Key words: flexion-relaxation, reflex, low back, spine, electromyogram, stability. **Spine 2006;31:839–845**

Trunk flexion postures are a well-recognized risk factor for low back pain (LBP).[1,2] Recent investigations have begun to link prolonged and/or cyclic trunk flexion to impaired neuromuscular function. Specifically, animal models show that the reflex response in the paraspinal muscles is disturbed following spine flexion.[3] This flexion-induced neuromuscular disturbance in those animals was attributed to creep deformation of the passive tissues in the spine.[4] Spinal creep deformation is also observed in humans following static flexion postures.[5] However, we are aware of no studies in human beings to investigate whether paraspinal reflexes are disturbed in association with spinal creep deformation from flexed torso postures.

Studies using a feline model show that extreme static or cyclic lumbar flexion causes neuromuscular dysfunction in the form of reduced muscle activity and reflexes. Paraspinal electromyographic (EMG) activity in these animals decreased significantly throughout 20 minutes of static and cyclic flexion.[3] During recovery, the animals showed initial transient hyper-excitability, followed by depressed reflex response for several hours, with slow recovery to normal. Measurements on human beings similarly showed hyper-excitable paraspinal reflex response immediately following static flexion.[6] Whether this behavior in human beings is transient and followed by reduced reflex response remains to be shown. Other joints have shown reduced reflex behavior in human beings following passive flexion.[7,8] No investigation of recovery time is available for human paraspinal reflexes.

One of the underlying mechanisms of reduced paraspinal reflexes associated with prolonged spinal flexion is linked to the function of the viscoelastic tissues in the spine.[9] Mechanoreceptors in the spinal ligaments reflexively activate the paraspinal musculature.[10–13] This reflex regulates continuous spinal movement, and acts to control stability to prevent damage from unexpected perturbations and extreme loads. During prolonged static and cyclic lumbar flexion, passive tissues in the spine may provide resistance against flexion load, allowing the trunk muscles to become deactivated.[14,15]

This phenomenon, known as flexion-relaxation, produces tissue laxity and creep, indicated by an increase in relative trunk angle over time.[5,16,17] Laxity in the viscoelastic tissues developed during extreme flexion may cause mechanoreceptors in the ligaments to become desensitized, reducing their ability to monitor vertebral movements and initiate reflexive muscular action.[4] Slow recovery of reflexes in a feline model may be caused by the slow recovery of viscoelastic tissue laxity.[18] Studies indicate that recovery from passive tissue creep in humans takes more time than is required to produce creep deformation (*i.e.*, recovery is slower than deformation).[5] Thus, human paraspinal reflexes should be expected to

From the Musculoskeletal Biomechanics Laboratories, Department of Engineering Science and Mechanics, School of Biomedical Engineering and Science, Virginia Polytechnic Institute and State University, Blacksburg, VA.
Acknowledgment date: October 27, 2004. First revision date: January 27, 2005. Second revision date: May 2, 2005. Acceptance date: May 2, 2005. The manuscript submitted does not contain information about medical device(s)/drug(s).
Federal funds were received in support of this work. No benefits in any form have been or will be received from a commercial party related directly or indirectly to the subject of this manuscript.
This research was supported by grants R01 AR46111 from NIAMS of the National Institute of Health and R01 OH07352-01 from NIOSH of the Centers of Disease Control.
Address correspondence and reprint requests to Kevin P. Granata, PhD, Musculoskeletal Biomechanics Laboratories, Department of Engineering Science and Mechanics, School of Biomedical Engineering and Science, VA Polytechnic Institute and State University, 219 Norris Hall (0219), Blacksburg, VA 24061; E-mail: Granata@VT.edu

show a similar pattern during recovery to that of the feline model.

Our previous studies observed modified paraspinal reflex following prolonged static flexion-relaxation[6] but did not record whether this effect was associated with postural creep deformation nor did it attempt to record recovery from the reflex disturbance. The goal of the current effort was to determine the effect of prolonged flexion-relaxation and recovery time on paraspinal reflex behavior in human subjects. We hypothesize that: (1) prolonged flexion-relaxation will result in reduced paraspinal reflex response; and (2) after a period of recovery equal in time to that of the flexion-relaxation, viscoelastic tissue creep and reflex response will not have fully recovered. We believe this will indicate that prolonged flexion-relaxation disturbs the neuromuscular system for a significant amount of time following flexion.

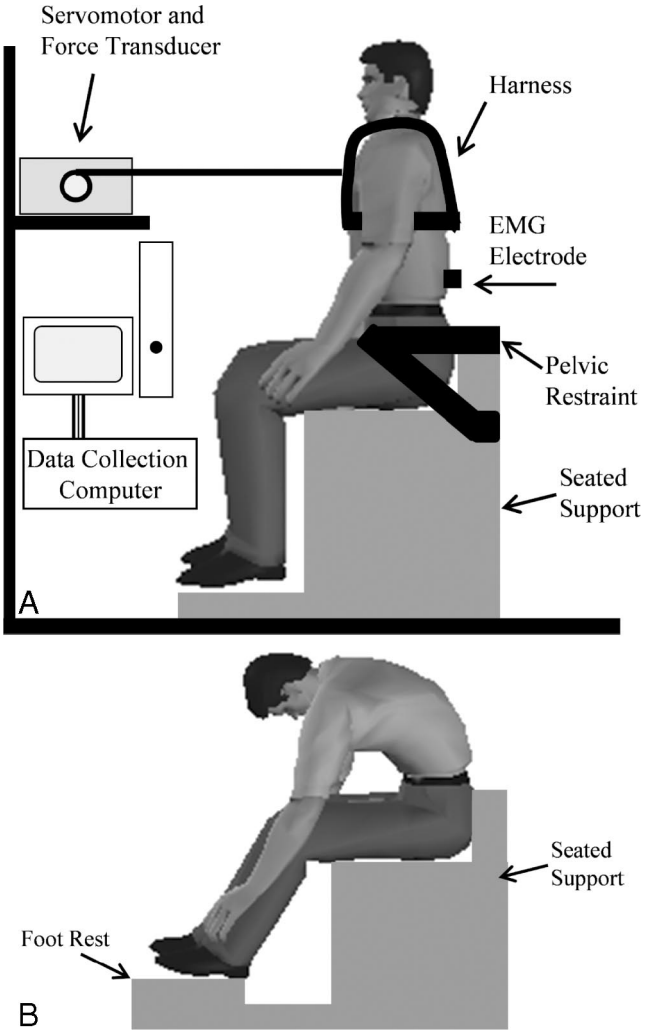

Figure 1. Subjects were seated in an upright posture for both reflex measurement and recovery (**A**). To quantify paraspinal reflex gain, a cable and harness system connected the subject to the servomotor, which applied pseudorandom force perturbations to elicit reflexes. To achieve passive spinal flexion, the subjects flexed forward into static flexion-relaxation (**B**). This posture was held for 4 cycles of 4 minutes each, separated by 1 minute of upright sitting. Reflexes were measured before and immediately after each static flexion-relaxation cycle.

■ Materials and Methods

Subjects. A total of 25 subjects with no previous history of LBP participated after signing informed consent approved by the institutional review board at Virginia Tech. Subjects had a mean age of 21.4 years (standard deviation [SD] 2.9). Subjects included 12 males with a mean height of 183.9 cm (SD 5.6) and a mean weight of 81.6 kg (SD 11.3), and 13 females with a mean height of 166.1 cm (SD 6.9) and a mean weight of 63.5 kg (SD 8.6).

Equipment. To test our hypotheses, lumbar paraspinal reflexes were examined before, during, and after prolonged flexion-relaxation, as well as during and after a recovery period. To record trunk muscle response and kinematic behavior, subjects were seated without trunk support in an upright posture (Figure 1A). A restraining belt immobilized their pelvis. A harness and cable system attached the subject to a servomotor such that cable tension applied flexion loads at the T10 level of the trunk. A servomotor (Pacific Scientific, Rockford, IL) was used to elicit paraspinal reflexes according to protocol described in our previous work.[6] The motor provided a constant isotonic preload of 100 N that the subjects were instructed to resist by maintaining their upright seated posture. Pseudorandom stochastic force perturbations of ±75 N were superimposed on the preload (Figure 2A). The applied forces were measured (Fig. 2B) by a force transducer attached to the motor (Omega, Stamford, CT) and EMG response to the force perturbations recorded from surface electrodes. For the subsequent protocol description, a reflex trial consists of 2, 10-second force perturbation trials of the servomotor, during which reflexes were elicited and recorded.

There were 3 surface-mounted, 6 degrees of freedom electromagnetic position sensors (Motion Star System, Ascension Technology Corp., Burlington, VT) placed on the subject at S1, T10, and the manubrium, and sampled at 100 Hz. Kinematic data were rectified and filtered using a 25 Hz, low-pass, seventh

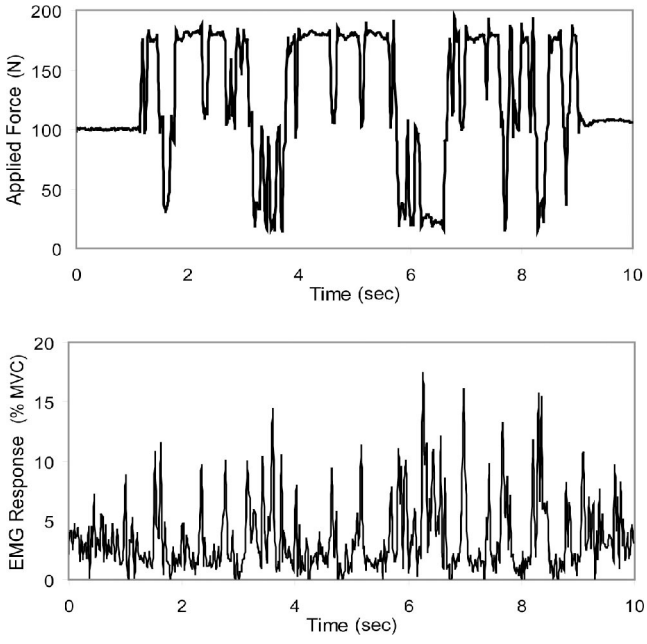

Figure 2. Applied force, including preload, and pseudorandom force perturbations (**A**). Rectified lumbar paraspinal muscle EMG data for a typical reflex trial (**B**).

order Butterworth filter in post-processing software (Matlab, Natick, MA). There were 8 pre-gelled active bipolar surface EMG electrodes placed on the subject at the left and right rectus abdominis, internal oblique, external oblique, and lumbar paraspinal muscles, with placement as described by Marras and Mirka.[19]

Data were collected using silver bar electrodes, with an interelectrode distance of 10 mm integrated into the active differential preamplifier (Delsys DE 2.1, Boston MA). A reference electrode was placed on the prominent bony section of the left tibia. EMG signals were amplified by 1000 with a frequency band-pass of 20–450 Hz, CMRR of 92 dB. EMG signals were recorded at 1000 Hz. EMG data were band-stop filtered at 60 Hz and 100 Hz in post-processing software to eliminate electromagnetic interference from the Motion Star System. EMG was rectified and low pass filtered at 25 Hz using a seventh order Butterworth filter. Both force and EMG signals were treated with the same filters to avoid phase shift discrepancy from processing. Rectified and filtered EMG was subsequently normalized to levels recorded during maximum voluntary exertions performed in isometric trunk flexion, extension, combined torsion and extension, and combined torsion and flexion.

Experimental Protocol. Experimental protocol was divided into 2 phases, static flexion-relaxation and recovery, each containing cycles. Paraspinal reflexes were recorded before the beginning of static flexion-relaxation, as described previously. To impose spinal ligament stretch, subjects remained within the pelvic restraint, which acted to prevent anterior tilt of the pelvis and initiated a full flexion posture by leaning forward (Figure 1B), as described by McGill and Brown,[5] and Solomonow *et al.*[20] Feet were positioned so that the knees were slightly bent and the amount of stretch occurring in the hamstrings was reduced. This posture was held for 4 minutes. Flexion-relaxation occurred while subjects were seated in full flexion, monitored by measured erector spinae EMG. The 4 periods of static flexion are referred to as static flexion-relaxation to indicate that subjects maintained full static flexion during which flexion-relaxation occurred.

During each cycle of static flexion-relaxation, both EMG and kinematic data were recorded for 5 seconds at the beginning of every minute. This process allowed us to measure the progression of trunk angle and monitor that erector-spinae EMG was in fact de-recruited to ensure flexion-relaxation. At the end of the 4-minute static flexion-relaxation period, subjects returned to an upright posture, and a reflex trial was recorded. This cycle was repeated 4 times, for a total of 16 minutes of static flexion-relaxation. Both the flexion-relaxation posture and reflex data collection were performed in a seated posture to facilitate transition time from flexion-relaxation to reflex measurement. Less than 1 minute was required between static flexion-relaxation cycles to record the reflex data and return them to the static flexion-relaxation posture.

Immediately following the static flexion-relaxation phase, subjects entered the recovery phase, which included 4, 4-minute cycles, each separated by a reflex trial. During each 4-minute recovery cycle, subjects sat in an upright and relaxed posture. At the beginning of each minute, they were instructed to resume the flexed posture for approximately 10 seconds so that kinematic data could be collected and recovery-progression of trunk angle could be monitored.

Although a reflex trial was recorded every 4 minutes and trunk angle every minute, only the 3 most relevant positions in time were analyzed statistically: time T_0 was the baseline measurement recorded before the first static flexion-relaxation cycle; time T_{FR} represents the measurement time immediately following the fourth and final cycle of static flexion-relaxation; time T_R was immediately following the fourth and final cycle of recovery.

Data Analysis. Reflex response was quantified in terms of latency and gain from the erector spinae EMG in the method of our previous work.[6] A nonparametric impulse response function (IRF) was calculated from the pseudorandom force input and the rectified EMG output of the erector spinae muscles. Calculation of the nonparametric IRF was based on deconvolution techniques for a time-delayed linear systems response.[21] For any linear time-invariant system, the output y(t) (*i.e.*, rectified EMG), was described by the IRF h(t) of the system convolved with (notated *) the applied input signal, x(t) (*i.e.*, external trunk force),

$$y(t) = \int_{T_1}^{T_2} h(\tau)x(t-\tau)dt = h(t) * x(t) \quad (1)$$

where the $h(\tau)$ is considered trivial for time lags $\tau < T_1$ and $\tau > T_2$. Using this property, any linear time-invariant system can be described in its entirety by its IRF because once h(t) is known, the output of the system can be determined for any input. Accordingly, the discrete-time IRF can be represented in terms of the input autocorrelation function and the input/output cross-correlation function:

$$c_{xy}(k) = \Delta t \sum_{j=M_1}^{M_2} h(j)c_{xx}(k-j) \quad (2)$$

where the input and output are sampled every Δt seconds, $M_1 = \frac{T_1}{\Delta t}$ and $M_2 = \frac{T_2}{\Delta t}$. This equation can then be written in matrix form as:

$$C_{xy} = \Delta t C_{xx} H \quad (3)$$

where C_{xy} is an $M_2 - M_1 + 1$ length vector whose i^{th} element is $c_{xy}(M_1 + i - 1)$, C_{xx} is an $M_2 - M_1 + 1$ square matrix whose i, j^{th} element is $c_{xx}(i - j)$ and H is an $M_2 - M_1 + 1$ length vector whose i^{th} element is $h(M_1 + i - 1)$. Using simple matrix inversion, one can solve for the IRF, H, representing the EMG response (Figure 3).

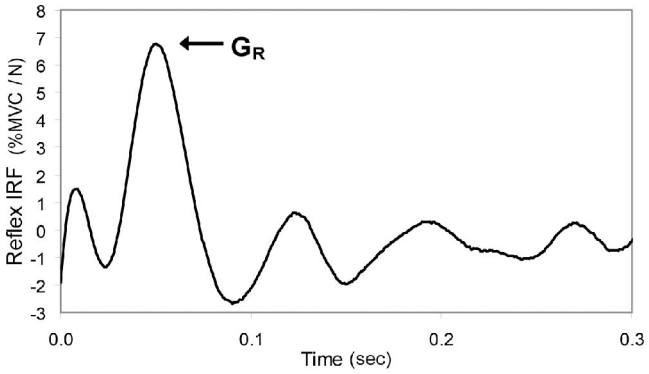

Figure 3. Typical IRF computed from applied trunk force (input) and rectified EMG (output). Reflex Gain, G_R, was competed from the peak of the IRF. Data at times longer than 120 milliseconds were assumed arbitrarily voluntary and, therefore, appears as noise in the IRF.

Reflex gain, G_R, represents the amplitude of the transfer function relating the paraspinal EMG response to the perturbation force (Figure 3). Reflex gain characterizes the magnitude of muscle reflex response scaled with respect to the time-dependent amplitude of the force disturbance. High G_R indicates a large reflex response, small G_R indicates minimal muscle activity following the disturbance. G_R was quantified based on the peak of the IRF computed from the applied input force and rectified EMG output of paraspinal muscles. Peak amplitude of the IRF must be higher than 2 SDs above the mean of the IRF to be considered a reflex. A second criterion required that G_R must occur between 20 and 120 milliseconds following the perturbation. This requirement ensured that the reflex response was not confounded by electromechanical delay or voluntary contribution to EMG response.

The dynamic trunk flexion-extension movement in response to the force perturbations was analyzed using an approach similar to that of the reflex gain. Movement was quantified by an IRF, using the applied force as the input and trunk movement (*i.e.*, linear position of T10 sensor) as the output response.[22] The kinematics gain, G_K, was estimated from the peak of the kinematics IRF (Figure 4).

To measure the quality of the IRF representation of both the reflex and kinematic responses, the computed IRF was convolved with the original pseudorandom force sequence to produce an estimate of the output signal. RMS difference between the physiologic signal and the estimated signal was recorded as percent variance accounted for (VAF). VAF equal to 100% indicates that the IRF exactly predicts the measured signal from the input force perturbations.

To obtain a measure of ligament elongation or laxity, 2 trunk angle measurements were recorded. The sagittal trunk angle, θ_T, recorded the overall angle of the trunk in the sagittal plane, determined from the difference in angle between the markers at S1 and the manubrium. The second measure was lordosis angle, θ_L, which was a relative angle of the curvature of the spine from S1 to T10. Lordosis angle was computed from the difference in the sagittal angle of the position sensors located at S1 and T10. Both trunk angle and lordosis angle were recorded while the subject was fully flexed. For comparison between subjects, trunk angle was taken as the difference from the first flexion angle recorded, *i.e.* angle of flexion-relaxation at T_0 was defined as zero. To quantify trunk muscle co-recruitment, the RMS level of the rectified, filtered, and normalized EMG signal from each muscle was quantified during the 250-millisecond period of constant isotonic load, immediately before initiation of the pseudorandom force perturbations.

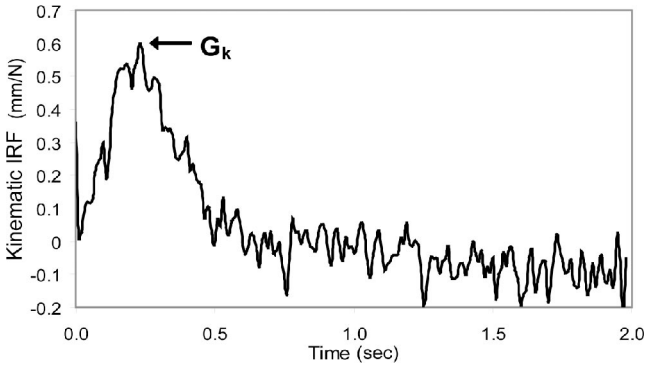

Figure 4. Typical impulse response function computed from input applied trunk force and output T10 displacement. Kinematics gain, G_K, is labeled.

Statistical Analysis. Statistical repeated measures analysis, analysis of variance, was performed to determine the effect of gender, static flexion-relaxation, and recovery on G_R, and G_K, trunk muscle recruitment, and trunk angle. The independent variable of gender was analyzed as a between subject variable, while the effects of static flexion-relaxation and recovery were analyzed as a within subject variable. Significance was determined at the level of $\alpha < 0.05$. Tukey honest-significant difference post hoc analyses were used to compare differences among significant treatments.

■ Results

Analyses revealed that the sagittal trunk angle, θ_T, increased significantly from T_0 to T_{FR} ($P < 0.001$) and from T_0 to T_R ($P < 0.001$), indicating tissue elongation occurred during the flexion protocol (Table 1). However, there was no significant change in θ_T from T_{FR} to T_R, possibly suggesting that the recovery phase of the protocol was insufficient to allow laxity in the passive tissues to recover. There was no significant difference in main effect of gender on θ_T.

Lordosis angle, θ_L, results also revealed increases caused by static flexion-relaxation from T_0 to T_{FR} ($P < 0.001$) and from T_0 to T_R, ($P < 0.001$). There was no significant change from T_{FR} to T_R, further indicating that passive tissues were unable to recover in this protocol. There was no significant main effect of gender on θ_L. Post hoc analyses of a significant gender-by-time interactions indicated that in males, there was a significant change in θ_L from T_0 to T_{FR} ($P < 0.02$) but not from T_0 to T_R ($P < 0.08$). However, in females, there was a significant increase in θ_L from T_0 to T_{FR} ($P < 0.001$) and T_0 to T_R

Table 1. Mean (SD) of Trunk Angle, Reflex Gain, and Kinematics Gain

	Men	Women	Average
θ_T (deg)			
T_0	0 (0)	0 (0)	0 (0)
T_{FR}	4.40 (4.99)*	6.11 (4.04)*	5.33 (4.47)†
T_R	5.65 (5.87)*	8.24 (5.28)*	7.06 (5.58)†
Average	3.35 (4.95)	4.28 (5.14)	4.13 (5.07)
θ_L (deg)			
T_0	0 (0)	0 (0)	0 (0)
T_{FR}	17.45 (16.16)*	22.25 (11.99)*	20.33 (13.61)†
T_R	14.20 (15.30)	23.70 (17.66)*	19.9 (17.02)†
Average	10.55 (14.51)	15.32 (16.26)	13.41 (15.63)
G_R			
T_0	4.09 (2.21)‡	6.03 (4.51)‡	5.06 (3.64)
T_{FR}	3.65 (2.71)	4.31 (2.20)*	3.98 (2.46)†
T_R	2.83 (1.57)	3.68 (2.37)*	3.25 (2.03)†
Average	3.52 (2.24)	4.67 (3.30)	4.10 (2.86)
G_K			
T_0	0.566 (0.097)	0.611 (0.104)	0.589 (0.097)
T_{FR}	0.492 (0.073)	0.581 (0.091)	0.539 (0.093)
T_R	0.543 (0.145)	0.586 (0.073)	0.565 (0.112)
Average	0.533 (0.107)	0.592 (0.088)	0.565 (0.101)

*Significance caused by flexion-relaxation within gender group ($P < 0.05$).
†Significant effect of flexion-relaxation ($P < 0.05$).
‡Significance caused by gender within time group ($P < 0.05$).

($P < 0.001$). There was no significant change from T_{FR} to T_R in males or females.

Minute-by-minute changes in θ_T and θ_L were analyzed during the flexion period to determine whether recovery took place during the minute of upright sitting required for reflex trials. The θ_T showed no significant recovery following the minute of upright sitting. However, θ_L did show significant recovery from the end of the 4-minute flexion periods to the beginning of the following flexion periods ($P < 0.008$). This recovery was significant only in females ($P < 0.022$).

Reflexes were observed in 84% of trials, with the remaining trials failing to satisfy the criteria for defining G_R. Reflex variance accounted for, VAF_{reflex}, had an average value of 47.4%. This average value of VAF_{reflex} is consistent with previous results in our laboratory.[6]

The main effect of reflex gain, G_R, caused by static flexion-relaxation was significant. The G_R decreased significantly from T_0 to T_{FR} ($P < 0.029$). There was a significant difference in G_R from T_0 to T_R, but no significant difference in G_R between T_{FR} and T_R, indicating that reflex gain failed to recover within the 16-minute recovery period. There was no significant main effect of gender on G_R. Post hoc analyses yielded significant differences in G_R between males and females at T_0. At T_0, females had significantly higher G_R than males ($P < 0.018$), but there was no difference between genders at T_{FR} or T_R. In males, neither static flexion-relaxation nor recovery was a significant factor on G_R. However, in females, reflex gain decreased significantly from T_0 to T_{FR} ($P < 0.047$) and from T_0 to T_R ($P < 0.001$). There was no significant difference in G_R between T_{FR} and T_R in either gender.

Movement analyses yielded an average value of kinematics variance accounted for, VAF_K, of 91.5%. Kinematics gain, G_K, showed a trend suggesting a decrease from T_0 to T_{FR} ($P < 0.062$) but no significant change from T_0 to T_R or T_{FR} to T_R. There was no significant main effect or interactions with gender in G_K.

The effects of gender, static flexion-relaxation, and recovery on trunk muscle recruitment were evaluated. No muscle groups showed significant gender differences in baseline EMG. Right and left external oblique muscles showed a significant decrease in baseline EMG activation level from T_0 to T_{FR}. There was no significant change from T_0 to T_R or from T_{FR} to T_R. No other muscle group showed significant differences in EMG activation level caused by static flexion-relaxation or recovery.

■ Discussion

Results show that prolonged static flexion-relaxation influenced the function of the neuromuscular system in human beings. Lumbar paraspinal reflex gain decreased significantly after 16 minutes of flexion-relaxation, which was consistent with our hypothesis. Reduced reflexes may suggest that the spine was less stable following prolonged flexion-relaxation, and, therefore, more susceptible to LBP and injury.[23] Research indicates that patients with LBP have reduced responsive control and delayed muscle responses compared to healthy subjects.[24,25] The decrease in reflex gain observed in the current study may be linked to the significant increase in spinal tissue laxity associated with static flexion-relaxation. An increase in trunk angle indicates that passive tissue creep occurred, and laxity in these tissues may have contributed to desensitization of neurosensors.[26]

However, the results of this investigation were inconsistent with our previous results where reflex behavior was recorded from subjects following 15 minutes of continuous static flexion-relaxation.[6] In that study, we found a trend toward increased reflex gain immediately following prolonged static flexion-relaxation, which we concluded was consistent with the hyper-excitable EMG response found in animal research.[26] There are several possible explanations as to why our previous results differ from what we found here. Solomonow *et al*[3] investigated muscle activity during both static and cyclic loading. In the case of static loading, hyper-excitability was present immediately following the end of loading, and EMG was typically increased from its baseline level. In the case of cyclic loading, the EMG magnitude following the loading was typically less than baseline. This result could be caused by the intermittent rest periods associated with cyclic loading, although further investigation into this issue is necessary. In the current study, subjects underwent 16 minutes of static flexion-relaxation, but the 4-minute cycles were separated by approximately 1 minute of seated upright active exertion to record reflexes (Figure 1). Therefore, our protocol may be considered cyclic rather than static flexion, and it may be reasonable to expect different results than static flexion-relaxation.

One major difference between this study and our previous investigation is subject position during reflex trials (*i.e.*, upright standing *vs.* upright sitting). It is possible that this difference may contribute to the discrepancy in our results regarding hyper-excitable *versus* depressed reflex, although it is unclear precisely what effect this difference has on reflex disturbance. One major drawback of our previous attempt to measure the effect of static flexion-relaxation on reflex response was the absence of trunk angle measurements during flexion. In the current study, the creep deformation of the trunk posture was documented to show that reflex change after static flexion-relaxation may be associated with the viscoelastic creep. Further research is necessary to determine whether this neuromechanical relationship is causative.

The second noteworthy result of this investigation was that reflex gain failed to change during the recovery phase. This result indicates that no appreciable recovery of paraspinal reflexes took place. This outcome may be because of the fact that neither lordosis nor sagittal trunk angle recovered within the experimental session, indicating that creep in the viscoelastic tissues may not have recovered sufficiently to restore sensitivity to the mechanoreceptors for reflex initiation. A 10-second period of flexion was performed each minute throughout the recovery phase to measure trunk angle and laxity recovery.

This repeated ligament strain may have been too frequent to allow recovery of passive tissues. The lack of reflex recovery may also be caused in part by the upright sitting used during the recovery phase because this posture in itself involves lumbar flexion.[27] Nevertheless, feline models showed depressed reflexes several hours after prolonged flexion.[28] Therefore, reflex disturbance may require longer to recover than 16 minutes following 16 minutes of static flexion-relaxation. Our results agree with the literature in that the recovery process for tissue creep and reflex response may be slow.

Of further interest is the progression of reflex gain and trunk angle throughout the entire protocol (Figure 5). Reflex gain was measured every 4 minutes during flexion-relaxation and every 4 minutes during recovery, for a total of 9 measurements. Trunk angle was measured every minute. Although only the main times of interest were statistically analyzed for changes in reflex, the patterns of change in lordosis angle and reflex are interesting to note. Following a cycle of static flexion-relaxation and 1 minute of sitting in an upright posture (*i.e.*, during reflex trials), θ_L showed significant recovery. Any recovery achieved was transient because by the second minute of the ensuing static flexion-relaxation cycle, θ_L returned to increased levels and continued increasing during the remaining static flexion-relaxation. This trend is interesting to note in conjunction with results from animal models showing that recovery may be transient.[4]

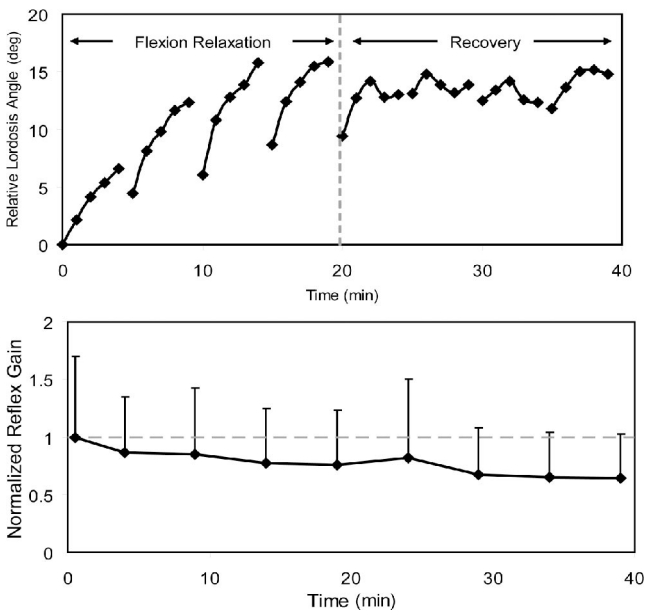

Figure 5. Progression of lordosis angle (**A**) and reflex gain (**B**). In the lordosis angle plot, the 4-minute cycles are depicted by connecting lines, whereas the minute in between cycles is represented by a disconnect. Significant recovery of lordosis angle was observed during the minute between trials, but the recovery was incomplete. Reflex gain was analyzed at time T_0 (baseline measurement recorded before the first static flexion-relaxation cycle), T_{FR} (immediately following the fourth and final cycle of static flexion-relaxation), and T_R (immediately following the fourth and final cycle of recovery), and revealed a significant decline following the static flexion-relaxation trials.

A 10-minute rest after 50 minutes of cyclic flexion loading of the feline lumbar spine allowed between 20% and 30% recovery of reflexive muscle activity. However, all recovery was completely lost after only 20 seconds of additional loading following the rest period. These results underscore the importance of adequate rest periods following torso flexion loading. One minute of rest after 4 minutes of flexion appeared to be inadequate for recovery of lumbar creep deformation and reflex gain. The importance of frequent and long rest periods during prolonged flexion is clear, but the amount of rest necessary to prevent significant reflex reduction and its influence on spinal stability has yet to be determined.

Lordosis angle results were consistent with McGill and Brown,[5] Dickey,[16] and Olson[17] *et al*, who showed that recovery of passive tissue creep takes longer than the time in which the creep occurred. During the recovery cycles, lordosis angle remained approximately constant, presumably because of the repeated bouts of flexion performed for data collection. However, θ_L clearly did not increase further, indicating that this duty cycle of flexion was not enough to cause further tissue creep. Further investigation into the disturbance of paraspinal reflexes as a result of various flexion duty cycles is necessary.

There was a trend suggesting reduced kinematic gain immediately following flexion-relaxation ($P < 0.062$). This finding is consistent with our previous results in which a significant decrease in kinematic gain was observed following 15 minutes of static flexion-relaxation. Decreased kinematic gain implies that trunk displacement decreased, and, therefore, trunk stiffness was increased following static flexion-relaxation. Active recruitment in the spinal musculature has increased trunk stiffness.[22] To reduce trunk displacement, spinal muscles should show increased activation levels following static flexion-relaxation.[29] However, of the 8 muscles examined, only the external oblique showed a significant change in baseline activity after static flexion-relaxation, and this was actually a reduction in activation after static flexion-relaxation. The other major muscle groups of the trunk showed no significant change after static flexion-relaxation, so increased trunk stiffness cannot be attributed to changes in cocontraction.

Andersson *et al*[30] showed that although superficial erector spinae muscles deactivate during flexion-relaxation, deep lumbar muscles, such as the quadratus lumborum and deep erector spinae, become more active with increasing trunk flexion angle. Measurement of these muscle groups would require indwelling EMG electrodes and, therefore, were not recorded in our study. It is possible that the activity of these muscles contributed to the reduced trunk displacement and apparent increase in stiffness following static flexion-relaxation.

Results of this study must be viewed in light of certain methodological limitations. The mean VAF_{reflex} of 47% agrees with published VAF values using similar linear EMG analyses.[31] However, nonlinear EMG analyses have yielded higher VAF values in other joints, so these

methods should be investigated in the future. An inherent limitation may occur from our protocol because of the fact that subjects were seated during reflex trials. Differences among results of this and previous studies may be influenced by the fact that upright sitting involves considerable flexion of the lumbar spine.[27] Loads imposed on laborers may be different from those in our experimental settings. The seated posture was used in this study to limit the transition time between static flexion-relaxation periods and reflex measurements. Trials discarded for not meeting the determined reflex gain criteria may indicate a limitation of the statistical analysis reported. Inclusion of these trials may have increased variability of results. Muscle fatigue may also influence the results, but because the paraspinal muscles were derecruited during the static flexion-relaxation postures, fatigue is highly unlikely. Despite these limitations, our results appear to correspond with literature concerning reflexes in a feline model.

Results confirm in human beings that prolonged static flexion results in passive tissue creep along with a neuromuscular disorder and that extended rest may be required for full recovery. Recovery of passive tissue creep and lumbar paraspinal reflexes required time longer than measured in this study, which was equal to the time spent in a flexed posture. Inhibited paraspinal reflexes may contribute to the risk of LBP or injury for workers using flexed postures, caused by the inability of the neuromuscular system to coordinate an appropriate muscle response following an unexpected loading event. General recommendations for occupational situations include limiting exposure to flexed posture work and providing long rest breaks. Further research is required for the recommendation of specific guidelines.

■ Key Points

- Reflexes in the paraspinal muscles and posture of lumbar flexion-relaxation were recorded in human subjects before and immediately after 16 minutes of cyclic flexion-relaxation tasks and after a recovery period.
- Trunk angle measured during flexion-relaxation postures was significantly higher following cyclic flexion-relaxation tasks, indicating creep deformation of passive supporting structures in the trunk.
- Reflexes were diminished following static flexion-relaxation tasks and failed to recover to baseline levels during 16 minutes of recovery.
- Results suggest that neuromuscular control of spinal stability may be limited following prolonged flexion-relaxation.

Acknowledgment

The authors thank M. Diersing, BS, and G. Slota, MS, for their assistance in data collection and processing.

References

1. Punnett L, Wegman DH. Work-related musculoskeletal disorders: The epidemiologic evidence and the debate. *J Electromyogr Kinesiol* 2004;14:13–23.
2. Marras WS, Lavender SA, Leurgans SE, et al. The role of dynamic three-dimensional trunk motion in occupationally-related low back disorders: The effects of workplace factors, trunk position and trunk motion characteristics on risk of injury. *Spine* 1993;18:617–28.
3. Solomonow M, Baratta RV, Zhou BH, et al. Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. *J Electromyogr Kinesiol* 2003;13:381–96.
4. Solomonow M, Zhou B, Baratta RV, et al. Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 1. Loss of reflexive muscular stabilization. *Spine* 1999;24:2426–34.
5. McGill SM, Brown S. Creep response of the lumbar spine to prolonged full flexion. *Clin Biomech (Bristol, Avon)* 1992;7:43–6.
6. Granata KP, Rogers E, Moorhouse KM. Effects of static flexion-relaxation on paraspinal reflex dynamics. *J Biomech* 2004;24:16–24.
7. Rosenbaum D, Hennig EM. The influence of stretching and warm-up exercises on Achilles tendon reflex activity. *J Sports Sci* 1995;13:481–90.
8. Avela J, Kyrolainen H, Komi PV. Altered reflex sensitivity after repeated and prolonged passive muscle stretching. *J Appl Physiol* 1999;86:1283–91.
9. Indahl A, Kaigle AM, Reikeras O, et al. Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. *Spine* 1997;22:2834–40.
10. Stubbs M, Harris M, Solomonow M, et al. Ligamento-muscular protective reflex in the lumbar spine of the feline. *J Electromyogr Kinesiol* 1998;8:197–204.
11. Panjabi MM. Clinical spinal instability and low back pain. *J Electromyogr Kinesiol* 2003;13:371–9.
12. Solomonow M, Zhou B, Harras M, et al. The ligamento-muscular stabilizing system of the spine. *Spine* 1998;23:2552–62.
13. Kang YM, Choi WS, Pickar JG. Electrophysiologic evidence for an intersegmental reflex pathway between lumbar paraspinal tissues. *Spine* 2002;27:E56–63.
14. Schultz AB, Haderspeck-Grib K, Sinkora G, et al. Quantitative studies of the flexion-relaxation phenomenon in the back muscles. *J Orthop Res* 1985;3:189–97.
15. Kippers V, Parker AW. Posture related to myoelectric silence of erectores spinae during trunk flexion. *Spine* 1984;9:740–5.
16. Dickey JP, McNorton S, Potvin JR. Repeated spinal flexion modulates the flexion-relaxation phenomenon. *Clin Biomech (Bristol, Avon)* 2003;18:783–9.
17. Olson MW, Li L, Solomonow M. Flexion-relaxation response to cyclic lumbar flexion. *Clin Biomech (Bristol, Avon)* 2004;19:769–76.
18. Gedalia U, Solomonow M, Zhou BH, et al. Biomechanics of increased exposure to lumbar injury caused by cyclic loading–Part 2. Recovery of reflexive muscular stability with rest. *Spine* 1999;24:2461–7.
19. Marras WS, Mirka GA. A comprehensive evaluation of trunk response to asymmetric trunk motion. *Spine* 1992;17:318–26.
20. Solomonow M, Baratta RV, Banks A, et al. Flexion-relaxation response to static lumbar flexion in males and females. *Clin Biomech (Bristol, Avon)* 2003;18:273–9.
21. Mirbagheri MM, Barbeau H, Kearney RE. Intrinsic and reflex contributions to human ankle stiffness: Variation with activation level and position. *Exp Brain Res* 2000;135:423–36.
22. Moorhouse KM, Granata KP. Trunk stiffness and dynamics during active extension exertions. *J Biomech* 2005;38:2000–7.
23. Panjabi MM. The stabilizing system of the spine. Part I Function, dysfunction, adaptation and enhancement. *J Spinal Disord* 1992;5:383–9.
24. Radebold A, Cholewicki J, Panjabi MM, et al. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. *Spine* 2000;25:947–54.
25. Radebold A, Cholewicki J, Polzhofer GA, et al. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. *Spine* 2001;26:724–30.
26. Solomonow M, Zhou BH, Baratta RV, et al. Biomechanics and electromyography of a cumulative lumbar disorder: Response to static flexion. *Clin Biomech (Bristol, Avon)* 2003;18:890–8.
27. Andersson GB, Murphy RW, Ortengren R, et al. The influence of backrest inclination and lumbar support on lumbar lordosis. *Spine* 1979;4:52–8.
28. Solomonow M, Zhou BH, Baratta RV, et al. Biexponential recovery model of lumbar viscoelastic laxity and reflexive muscular activity after prolonged cyclic loading. *Clin Biomech (Bristol, Avon)* 2000;15:167–75.
29. Cholewicki J, Juluru K, McGill SM. Intra-abdominal pressure mechanism for stabilizing the lumbar spine. *J Biomech* 1999;32:13–7.
30. Andersson EA, Oddsson LIE, Grundstrom H, et al. EMG activities of the quadratus lumborum and erector spinae muscles during flexion-relaxation and other motor tasks. *Clin Biomech (Bristol, Avon)* 1996;11:392–400.
31. Kearney RE, Hunter IW. System identification of human triceps surae stretch reflex dynamics. *Exp Brain Res* 1983;51:117–27.