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Effects of Static Flexion-relaxation on Paraspinal Reflex Behavior

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Abstract

Background.—Static trunk flexion working postures and disturbed trunk muscle reflexes are related to increased risk of low-back pain. Animal studies conclude that these factors may be related; passive tissue strain in spinal ligaments causes subsequent short-term changes in reflex. Although studies have documented changes in the myoelectric onset angle of flexion-relaxation following prolonged static flexion and cyclic flexion we could find no published evidence related to the human reflex response of the trunk extensor muscles following a period of static flexion-relaxation loading.

Methods.—Eighteen subjects maintained static lumbar flexion for 15 min. Paraspinal muscle reflexes were elicited both before and after the flexion-relaxation protocol using pseudorandom stochastic force disturbances while recording EMG. Reflex gain was computed from the peak value of the impulse response function relating input force perturbation to EMG response using time-domain deconvolution analyses.

Findings.—Reflexes showed a trend toward increased gain after the period of flexion-relaxation ($P < 0.055$) and were increased with trunk extension exertion ($P < 0.021$). Significant gender differences in reflex gain were observed ($P < 0.01$).

Interpretations.—Occupational activities requiring extended periods of trunk flexion contribute to changes in reflex behavior of the paraspinal muscles. Results suggest potential mechanisms by which flexed posture work may contribute to low-back pain. Significant gender differences indicate risk analyses should consider personal factors when considering neuromuscular behavior.

Keywords

Low-back; Reflex; Flexion-relaxation

1. Introduction

Workers who undergo periods of static or cyclic trunk flexion have an increased risk of low-back pain (Marras et al., 1993; Punnett et al., 1991). Biomechanical factors contributing to this risk may include excessive or cumulative spinal load (Kerr et al., 2001), ligament and disc strain (Dolan et al., 1994) and spinal instability (Omino and Hayashi, 1992). Active muscular recruitment and reflexes play a major role in both spinal and ligamentous load as well as spinal stability (GardnerMorse and Stokes, 1998; Granata and Marras, 1995). However, neuromuscular activity may be disturbed by periods of static or cyclic strain in the spinal ligaments and discs (Jackson et al., 2001; Solomonow et al., 1999). A change in reflex response associated with flexed working postures may contribute to limitations in stabilizing control and risk of injury (Solomonow et al., 1998). The goal of this study was to quantify changes in reflex response associated with prolonged trunk flexion in healthy human subjects.

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Flexed trunk postures can apply tensile strain to paraspinal muscles and passive viscoelastic tissues of the spine. Flexion-relaxation occurs during extreme lumbar flexion wherein strain of the passive tissues in the trunk and spine support the external flexion load thereby allowing deactivation of the trunk muscles (Kippers and Parker, 1984). Prolonged passive stretch of skeletal muscles influences gamma motor-neuron drive to reduce muscle spindle excitability. Studies in the ankle joint document reflex inhibition following static and cyclic stretch loading (Avela et al., 1999; Rosenbaum and Hennig, 1995). Flexion strain in the ligaments may also influence reflex behavior. Flexion loading of the ligamentous tissues of the spine causes intervertebral laxity; specifically tissue creep and stress-relaxation. These effects have been quantified in human cadaveric tissue (Twomey and Taylor, 1982), animal models (Solomonow et al., 2001), and in vivo human measurements (McGill and Brown, 1992). Existence of reflex pathways have been observed originating from spinal ligaments to surrounding muscles; specifically the multifidus and longissimus (Kang et al., 2002). Thus, a perturbation force applied to the trunk causes transient tissue strain and excitation of large mechanoreceptors (Golgi, Pacinin, Ruffini fibers) in the spinal ligaments as well as type Ia and type II afferent muscle spindle sensors to initiate reflexive action of the paraspinal muscles (Simon, 1994). Biomechanical laxity of the spine from prolonged trunk flexion may influence neurosensor strain and reflex in response to force perturbations (Jackson et al., 2001). Therefore, changes in reflex response following prolonged strain of the spinal ligaments may be related in part to ligament laxity (Solomonow et al., 1999). Although studies have documented changes in the myoelectric onset angle of flexion-relaxation following prolonged static flexion and cyclic flexion (Dickey et al., 2003; Solomonow et al., 2003a), we could find no published evidence related to the human reflex response of the trunk extensor muscles following prolonged passive stretch.

To date, studies linking reflex disturbances to prolonged spinal flexion are limited primarily to animal models (Claude et al., 2003). In surgical preparations a mechanical strain was applied to feline supraspinal ligaments and the electromyographic response was observed in adjacent paraspinal muscles (Solomonow et al., 1998). Following repeated ligament strain a brief period of reflex hyper-excitability was observed (Jackson et al., 2001). However, this period of hyper-excitability was short lived. Ligament laxity caused the mechanoreceptors to become desensitized thereby inhibiting electromyographic response of the paraspinal muscles for many hours (Gedalia et al., 1999). This laxity and disturbed reflex behavior may influence spinal stability (McGill and Brown, 1992). However, we could find no studies that validate these trends in humans. It is necessary to establish whether similar effects are observed from flexion-relaxation postures in humans.

To quantify changes in reflex response, systems identification techniques can be applied to determine the myoelectric response attributable to a force perturbation, i.e. reflex amplitude is represented in terms of the amplitude and temporal characteristics of the impulse response (Zhang et al., 1999). This measurement technique was applied to quantify the reflex response from measured electromyography (EMG) from the paraspinals before and after a period of static flexion-relaxation in healthy human subjects. Based on the available literature we hypothesized that prolonged flexion-relaxation in healthy subjects influences paraspinal reflex response behavior.

2. Methods

2.1. Subjects

Eighteen subjects, with no previous history of low-back pain participated after signing informed consent approved by the institutional review board at Virginia Tech. Subjects included nine females with mean (standard deviation) height 164.8 (7.2) cm and weight 61.1 (9.6) kg, and nine males with mean height 183.1 (7.4) cm and mean weight 75.9 (9.2) kg.

2.2. Protocol

Muscle recruitment, reflex gain, and perturbation kinematics were quantified before and immediately following a 15-min period of static passive flexion loading of the spine. To impose spinal ligament strain subjects sat on the floor with their trunk in full lumbar flexion and legs extended with a small padded bolster under the knees to reduce hamstring stretch and to tilt the pelvis posteriorly. They relaxed in this flexed posture while observing EMG from the erector spinae displayed on an oscilloscope to assure voluntary de-recruitment of the erector spinae, i.e. flexion-relaxation. Immediately following the flexion-relaxation period neuromechanical measurements of muscle recruitment, reflex gain and perturbation kinematics were repeated.

To record muscle response and kinematic behavior subjects stood upright with their pelvis and legs strapped to a rigid support (Fig. 1). A harness and cable system attached the subject to a servomotor (Pacific Scientific, Rockford, IL, USA) such that cable tension applied flexion loads at the T10 level of the trunk. The motor was programmed to provide three levels of isotonic load, 100 N, 135 N, and 170 N. Superimposed on the preload were force perturbations of ± 75 N applied in a pseudorandom stochastic fashion (Fig. 2a) with a flat bandwidth from 0-10 Hz. Pseudorandom stochastic inputs were chosen to avoid voluntary responses from predictable stimuli (Kearney and Hunter, 1990). The applied forces were measured by a force transducer attached to the motor sampled at 1000 Hz (Omega, Stamford, CT, USA). Three pseudorandom perturbation trials of 10 s each were performed at each preload level presented in random order before and after the flexion-relaxation protocol. After flexion-relaxation, some recovery may have occurred during the data collection trials. Statistical order effects were minimized by the trial randomization. Furthermore, only 3 min were required for data collection which was well below the documented recovery time constant, 9.4 min, reported by McGill and Brown (1992).

EMG was recorded from bipolar surface EMG electrodes (Delsys, Boston, MA, USA) on the left and right rectus abdominus, internal oblique, external oblique, and erector spinae with placement as described by Marras (Marras and Mirka, 1992). EMG data were bandpass filtered in hardware between 20 and 450 Hz and sampled at 1000 Hz. The EMG signals were rectified and filtered using a 25 Hz, low-pass, seventh-order Butterworth filter in post-processing software (Fig. 2b). Measured EMG data were normalized with respect to maximum voluntary exertion (MVE) in isometric trunk flexion, extension, combined torsion and extension, and combined torsion and flexion, recorded prior to the experiment. Trunk kinematics in response to the perturbation forces were recorded using surface mounted infrared motion sensors (Northern Digital, Waterloo, Ontario, Canada). Sensors were placed over the subject's spinous processes at S1, L5, T10 and C7 and sampled at 100 Hz (Fig. 2c). Kinematic data were filtered using a 25 Hz, low-pass, seventh-order Butterworth filter in post-processing software (Matlab, Natick, MA, USA).

2.3. Analysis

Reflex response was quantified in terms of latency and gain from the erector spinae EMG. 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 (Mirbagheri et al., 2000). For any linear time-invariant (LTI) system, the output $y(t)$, i.e., rectified EMG, was described by the impulse response function $h(t)$ of the system convolved with (notated) the applied input signal, $x(t)$, i.e. external trunk force,

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

where the $h(s)$ is considered trivial for time lags $s < T_1$ and $s > T_2$. Using this property, any LTI 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 impulse response function 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 s, $M_1 = \frac{T_1}{\Delta t}$ 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 impulse response function, H , representing the EMG response (Fig. 3). After $T_2 = 120$ ms any response is considered to be arbitrarily voluntary and therefore not quantified as a reflex (Matthews, 1991).

Both the applied trunk force and the rectified EMG were scaled to yield a mean value of zero prior to analysis in order to ensure accuracy of correlation function estimations. Published techniques illustrate that adding white noise to the input signal reduces the effect of system noise by decorrelating the data sampling artifact from the input signal (Ljung and Ljung, 1998). The magnitude of white noise added was 30% of one standard deviation of the input trunk force.

To measure the quality of the IRF representation of the reflex response, the computed IRF was convolved with the original pseudorandom force sequence to produce an estimate of the EMG signal. Root-mean-square (RMS) difference between the physiologic signal and the estimated signal was recorded as percent variance accounted for (VAF_{Reflex}) (Hunter and Kearney, 1983). VAF_{Reflex} equal to 100% indicates that the IRF predicts exactly the measured EMG signal from the input force perturbations. Reflex gain, G_R , was determined from the peak amplitude of the IRF and the latency, t_R , was the time at which the maximum peak occurred (Fig. 3). For this analysis a reflex response was defined as a peak occurring two standard deviations or more above the mean IRF signal. If no peak was above this threshold the trial was discarded.

Since perturbation forces were applied in a random time sequence the trunk movement had random qualitative appearance (Fig. 2c). To quantify movement amplitude kinematic gain, G_K , was estimated from the peak of the IRF determined from the pseudorandom force input and the position output recorded from the infrared motion sensors (Fig. 4). G_K represents the dynamic trunk flexion-extension movement in response to the force perturbations (Moorhouse and Granata, in press). Analogous to the reflex data, VAF_K was computed to estimate the quality of the kinematic measurement.

Secondary analyses included quantification of baseline trunk muscle recruitment and pelvic angle. To quantify trunk muscle recruitment the RMS level of the normalized, rectified and filtered EMG signal from each muscle was quantified during the period of constant isotonic load, i.e. 250 ms mean value recorded immediately before the initiation of the pseudorandom force perturbations. Although subjects maintained an upright trunk posture lumbar lordosis was not controlled. Pelvic angle was quantified as an analog to lordosis and measured from

the angle between the two kinematic sensors placed at S1 and L5. This was recorded during the period of constant isotonic load prior to initiation of the pseudorandom force perturbations of each trial.

2.4. Statistical analysis

Statistical repeated measures analyses (ANOVA) were performed to determine the effect of flexion-relaxation, gender and preload on baseline EMG activity, G_R , t_R , $VA_{F_{Reflex}}$, G_K , and VA_{F_K} . The independent variable of gender was analyzed as a between subject variable while flexion-relaxation effect and preload were analyzed as within subject variables. 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. To quantify representative changes in antagonistic coactivation and lumbar lordosis the RMS EMG activity from the rectus abdominis and pelvic angle were similarly tested for influence of gender and flexion-relaxation.

3. Results

Reflexes were observed in 81.4% of the trials. There was no significant difference in rate of reflex occurrence for gender, flexion-relaxation effect or preload. $VA_{F_{Reflex}}$ had an average value of 48%. There were no significant main effects of gender, flexion-relaxation, or preload on $VA_{F_{Reflex}}$. However, the effect of preload approached significance ($P = 0.056$). This phenomenon is attributed to the fact that at higher preload levels EMG response is greater, resulting in a higher signal to noise ratio. At lower preloads the lower signal to noise ratio negatively affects the accuracy of the impulse response. VA_{F_K} had an average value of 93%. There were no significant effects of flexion-relaxation, preload level, or gender on VA_{F_K} .

Analyses revealed that reflex gains, G_R , were influenced significantly by flexion-relaxation, gender and preload as well as a significant gender-by-preload interaction (Tables 1 and 2). Three way interactions yielded no significance effects and therefore were not reported. Females had larger reflex G_R than males ($P < 0.01$). Post hoc analyses of the gender-by-flexion-relaxation interaction revealed that G_R was greater in females than in males after the flexion-relaxation protocol ($P < 0.0002$) but the gender difference failed to reach statistical significance before the period of flexion-relaxation ($P = 0.19$).

The main effect of flexion-relaxation in reflex gain was slightly outside the range of significance ($P = 0.055$) with a trend suggesting an increase in G_R after flexion. Post hoc analyses indicated a significant increase in G_R after flexion-relaxation ($P < 0.003$) in females. Males had no significant change for the number of subjects tested.

Reflex gain increased with preload. A significant difference in G_R occurred between the lowest and highest preload settings ($P < 0.02$), with reflex gain, G_R , being greater at higher preloads. There was also a significant gender difference in G_R at each preload level, with G_R being greater in women than men. There was no significant difference in reflex latency, t_R , due to preload, gender, or flexion-relaxation protocol.

There was a significant main effect of flexion-relaxation in kinematic gain with G_K being reduced after flexion-relaxation ($P < 0.003$). There was no significant difference in G_K between males and females. However, gender-by-flexion-relaxation interactions revealed a significant effect due to flexion-relaxation in females ($P < 0.011$). There was no significant effect of flexion-relaxation in males for G_K ($P = 0.72$). There was a significant main effect of preload on G_K , with the highest G_K occurring at the lowest preload.

Effects of gender and flexion-relaxation on baseline muscle activity and pelvic angle were evaluated (Table 3). Rectus abdominis coactivation was greater in females than in males both before and after flexion-relaxation ($P < 0.001$). In females the baseline rectus abdominis coactivation showed no significant change due to flexion-relaxation. In males, there was a trend toward increased coactivation following flexion-relaxation ($P = 0.070$). Pelvic angle increased following flexion relaxation ($P < 0.002$). A significant gender-by-flexion relaxation interaction revealed that the change was significant in males ($P < 0.024$), but not in female participants ($P = 0.23$). There was no main effect of gender in pelvic angle.

4. Discussion

Four primary effects were observed in the results. First, trends indicated higher reflex gain following a period of static flexion-relaxation, specifically in females. This indicates greater EMG response per unit of perturbation force, i.e. hyper-excitable paraspinal reflex response following 15 min of flexion-relaxation. Second, reflex gain increased with trunk flexion preload. Third, females exhibited larger reflex gains than males. Finally, kinematic gain decreased following flexion-relaxation indicating less trunk movement in response to a force perturbation.

Matthews (1991) showed that the human stretch reflex consists of at least two different components including a short latency spinal reflex and a long latency transcortical reflex. The short latency reflex, occurring between 10 and 30 ms was observed in some trials (Fig. 3) but the amplitude was smaller than the peak G_R and therefore not included in the statistical analyses. Peak amplitude of the long latency reflex was represented by the reflex gain, G_R , of the IRF and typically occurs around 60 ms (Matthews, 1991). This accounts for conduction time from the spindle and mechanoreceptors, through a multisynaptic reflex loop and back to the muscle as well as propagation time of the muscle action potentials. Magnusson et al. (1996) noted that sudden flexion loading and unloading of the trunk yielded mean reflex delays of 100 ms and 50 ms respectively. Thus, experimental protocol may be an important factor when examining reflex latency. In the current study the perturbation loading was stochastic and included both flexion loading and flexion unloading with mean reflex latency $t_R = 67$ ms. Reflex latency, t_R , showed no significant effects for gender, flexion-relaxation or preload level. However, the response rate was fast enough that it may contribute to control of the trunk in response to external force disturbances.

Trends toward increased reflex gain, G_R , were observed following 15 min of static flexion-relaxation posture ($P = 0.055$). Passive stretch of skeletal muscles can reduce muscle spindle excitability thereby inhibiting reflex amplitude (Avela et al., 1999; Rosenbaum and Hennig, 1995). Conversely, animal models demonstrate that passive stretch of spinal ligaments can cause brief hyper-excitability of the paraspinal muscle response (Solomonow et al., 2003b). Our results suggest similar behavior is observed in humans. The force sequences before and after the flexion-relaxation protocol were similar in amplitude and signal character. However, this does not guarantee similar movement dynamics. Kinematic response to the force perturbations, G_K , was reduced following 15 min of static flexion-relaxation. Hence, the force disturbance caused less trunk movement after the flexion-relaxation protocol than before. This was contrary to our expectations because published data suggest reduction in stiffness following prolonged flexion-relaxation (McGill and Brown, 1992; Solomonow et al., 2003a) suggesting increases in G_K should be expected. An increase in baseline muscle contraction could partially explain the reduced G_K values after flexion-relaxation. However, no changes in baseline EMG levels were observed in the data. It should be noted that a change in G_K was significant only in the female participants. Similarly, the increase in reflex gain, G_R , was significant only in the female participants. Thus, we believe that the decrease in movement following flexion-relaxation may be attributed to the increase in reflex gain. Recognizing that

the natural kinematic frequency of the trunk is approximately 1 Hz (Fig. 4) the reflex response can contribute as feedback control to limit movement following an external force disturbance. This suggests that paraspinal reflexes may play a significant role in the control of trunk muscle stiffness and associated spinal stability. In animal models brief periods of hyperexcitable reflexes after flexion-relaxation was followed by prolonged periods of reflex depression that may limit spinal stability (Claude et al., 2003; Jackson et al., 2001; Solomonow et al., 1999). Further research is necessary in human subjects to determine whether reflex inhibition is observed following the period of hyper-excitability associated with prolonged flexion relaxation.

Reflex gain increased with trunk extension effort. Motor neurons that initiate the reflex action are influenced by muscle tone and may not be excited if the muscle is completely relaxed (Matthews, 1986). Therefore, research shows that reflex excitation increases with low-level preactivation of muscles whereas at high exertion levels muscle stiffness is increased thereby causing reduced displacement and reflex response (Bennett et al., 1994; Matthews, 1986). The saturation point between enhanced reflex excitability and reduced response is generally considered to be approximately 50% of maximum voluntary contraction. During trunk extension exertions Stokes et al. (2000) showed that muscle responses occurred less frequently at high preloads than at low exertion levels. In the current study, mean distance from the L5/S1 junction to the level of T10 where the force perturbation was applied was 14.9 cm indicating that the three preloads were equivalent to external flexion moments of 14.9 N m, 20.1 N m, and 25.3 N m. These are less than 20% of the maximum extension moment strength reported by Parnianpour (Parnianpour et al., 1991). Because the effort levels were below 50% MVC, one should expect increased reflex gain with increased preload. Thus, results were consistent with the neurophysiologic literature, i.e. for the three preload levels tested reflex gain increased significantly from the lowest to the highest level.

Reflex gain was greater in females than males. Since force perturbations of 75 N were applied to both males and females alike, one might expect that the smaller trunk inertia of the women would result in greater movement and subsequently larger G_K . This was not observed. However, we have noted elsewhere that trunk stiffness is greater in females than in males (Moorhouse and Granata, in press). Increased stiffness combined with smaller inertia causes high natural frequency of motion indicating faster movement in females. The reflex response is strongly influenced by tissue strain rate (Simon, 1994). An estimate of the measured EMG response was achieved by convolving the reflex IRF with the measured input force signal for comparison with the actual EMG signal. The variance accounted for (VAF) by the IRF explained approximately half of the EMG signal in the current study. This demonstrates the ability to identify reflex behavior from force perturbations. It also highlights a limitation of the study, i.e. further efforts must attempt to reduce the variability of the reflex gain estimate. Mirbagheri et al. (2000) concluded that VAF of the reflex response may exceed 95% if one uses kinematic velocity as the reference disturbance rather than the external force signal examined in the current study. Hence, future research must quantify the paraspinal muscle reflex response with respect to trunk kinematics. Nonetheless, if the force disturbance produced faster initial kinematic disturbances in the female participants then greater reflex gain should be expected. This has been observed in the knee joint and may explain the gender-specific behavior observed in our data (Shultz et al., 2004). We believe this velocity dependent contribution to increased reflex gain in women helped to limit movement amplitude following the external force disturbances and explains why G_K was similar in men and women. Further study is necessary to understand gender-specific neuromuscular control behaviors that may influence spinal stability.

Several effects remain unexplained. Why was the change in reflex gain following flexion-relaxation observed only in females? As noted above, we believe a gender difference in trunk

stiffness may contribute to reflex gain but this fails to explain why flexion-relaxation caused a change in neither G_R nor G_K in male subjects. It is possible that mechanical and neuromuscular response to flexion-relaxation postures is gender specific. Published studies indicate that females developed more spinal laxity than males during static flexion-relaxation protocols (McGill and Brown, 1992; Solomonow et al., 2003a). Nonetheless, those studies report significant increases in trunk laxity and associated tissue creep in males. Hence, we expected change in reflex in the males as well as in females. Trunk muscle co-contraction may contribute to gender effects. Our results agree with previous measurements wherein gender differences in antagonistic coactivity of the trunk muscle have been observed (Granata et al., 2001). There was no change in baseline EMG following flexion-relaxation in either males or females (Table 3) but the male participants demonstrated a trend toward greater antagonistic coactivation after flexion-relaxation than before ($P < 0.07$). Spinal posture may also influence reflex gain by modulating the muscles and ligament strain lengths. Although all subjects maintained an upright trunk posture both before and after the flexion-relaxation protocol, pelvic tilt angle increased following flexion relaxation in male subjects suggesting greater lumbar lordosis. No change in posture was observed in the female subjects. The combined effects of co-contraction and spinal curvature may influence gender-specific response to flexion-relaxation postures. Gender differences in reflex response to the flexion-relaxation protocol may suggest complex neuromotor and biomechanical mechanisms, and may indicate potential gender differences to injury risk from static flexion work postures.

In conclusion, paraspinal reflexes demonstrated a trend toward increased reflex gain immediately following 15 min of static flexion-relaxation posture. These results agree with animal models wherein a hyper-excitable reflex response is observed following prolonged ligament stretch (Solomonow et al., 2003c). It is noteworthy that those feline models demonstrate that the period of hyper-excitability is brief and followed by a prolonged reflex depression lasting up to 7 h. The current study did not record the recovery of the reflex gain so it remains to be demonstrated whether reflex depression is observed in humans following flexion-relaxation. These changes in reflex response may influence stability control and risk of low-back pain. Patients with lowback pain demonstrate reduced and slowed reflexes compared to asymptomatic control subjects (Luoto et al., 1996; Radebold et al., 2000) but it remains unclear whether this is a compensatory consequence of low-back pain (Van Dieen et al., 2003) or a contributing cause. Nonetheless, it has been proposed that changes in paraspinal muscle response following flexed posture work may contribute to LBP risk (Holm et al., 2002). Results suggest that occupational activities that require prolonged trunk flexion may contribute to changes in reflex behavior of the paraspinal muscles. Results also indicate that risk analyses must consider personal factors including gender when considering neuromuscular behavior that influence spinal load and stability.

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References

- Avela J, Kyrolainen H, Komi PV. Altered reflex sensitivity after repeated and prolonged passive muscle stretching. *J. Appl. Physiol* 1999;86:1283–1291. [PubMed: 10194214]
- Bennett DJ, Gorassini M, Prochazka A. Catching a ball: contributions of intrinsic muscle stiffness, reflexes, and higher order responses. *Can. J. Physiol. Pharmacol* 1994;72:525–534. [PubMed: 7954082]
- Claude LN, Solomonow M, Zhou BH, Baratta ERV, Ping M. Neuromuscular dysfunction elicited by cyclic lumbar flexion. *Muscle Nerve* 2003;27:348–358. [PubMed: 12635122]

- Dickey JP, McNorton S, Potvin JR. Repeated spinal flexion modulates the flexion-relaxation phenomenon. *Clin. Biomech* 2003;18:783–789.
- Dolan P, Earley M, Adams MA. Bending and compressive stresses acting on the lumbar spine during lifting activities. *J. Biomech* 1994;27:1237–1248. [PubMed: 7962011]
- Gardner-Morse MG, Stokes IAF. The effects of abdominal muscle coactivation on lumbar spine stability. *Spine* 1998;23:86–92. [PubMed: 9460158]
- Gedalia U, Solomonow M, Zhou BH, Baratta RV, Lu Y, Harris M. Biomechanics of increased exposure to lumbar injury caused by cyclic loading--Part 2. Recovery of reflexive muscular stability with rest. *Spine* 1999;24:2461–2467. [PubMed: 10626308]
- Granata KP, Marras WS. An EMG assisted model of biomechanical trunk loading during free-dynamic lifting. *J. Biomech* 1995;28:1309–1317. [PubMed: 8522544]
- Granata KP, Orishimo K, Sanford AH. Trunk muscle coactivation in preparation for sudden load. *J. Electromyogr. Kinesiol* 2001;11:247–254. [PubMed: 11532595]
- Holm SH, Indahl A, Solomonow M. Sensorimotor control of the spine. *J. Electromyogr. Kinesiol* 2002;12:219–234. [PubMed: 12086817]
- Hunter IW, Kearney RE. Two-sided linear filter identification. *Med. Biol. Eng. Comput* 1983;21:203–209. [PubMed: 6887997]
- Jackson M, Solomonow M, Zhou B, Baratta RV, Harras M. Multifidus EMG and tension-relaxation recovery after prolonged static lumbar flexion. *Spine* 2001;26:715–723. [PubMed: 11295887]
- Kang YM, Choi WS, Pickar JG. Electrophysiologic evidence for an intersegmental reflex pathway between lumbar paraspinal tissues. *Spine* 2002;27:E56–E63. [PubMed: 11805709]
- Kearney RE, Hunter IW. System identification of human joint dynamics. *Crit. Rev. Biomed. Eng* 1990;18:55–87. [PubMed: 2204515]
- Kerr M, Frank J, Harry S, Norman RW, Wells R, Neumann P, Claire B. Biomechanical and psychophysical risk factors for low-back pain at work. *Am. J. Pub. Health* 2001;91:1069–1075. [PubMed: 11441733]
- Kippers V, Parker AW. Posture related to myoelectric silence of erectors spinae during trunk flexion. *Spine* 1984;9:740–745. [PubMed: 6505844]
- Ljung, L.; Ljung, EJ. *System Identification: Theory for the User*. second ed.. Upper Saddle River, Prentice Hall; NJ: 1998.
- Luoto S, Taimela S, Hurri A, Aalto H, Pyykko I, Alaranta H. Psychomotor speed and postural control in chronic low back pain patients. A controlled follow-up study. *Spine* 1996;21:2621–2627. [PubMed: 8961450]
- Magnusson ML, Aleksiev A, Wilder DG, Pope MH, Spratt K, Lee SH, Goel VK, Weinstein JN. Unexpected load and asymmetric posture as etiologic factors in low back pain. *Eur. Spine J* 1996;5:23–35. [PubMed: 8689414]
- Marras WS, Mirka GA. A comprehensive evaluation of trunk response to asymmetric trunk motion. *Spine* 1992;17:318–326. [PubMed: 1533061]
- Marras WS, Lavender SA, Leurgans SE. 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;18:617–628. [PubMed: 8484154]
- Matthews PB. The human stretch reflex and the motor cortex. *Trends Neurosci* 1991;14:87–91. [PubMed: 1709536]
- Matthews PBC. Observations on the automatic compensation of reflex gain on varying the pre-existing level of motor discharge in man. *J. Physiol* 1986;374:73–90. [PubMed: 3746703]
- McGill SM, Brown S. Creep response of the lumbar spine to prolonged full flexion. *Clin. Biomech* 1992;7:43–46.
- Mirbagheri MM, Barbeau H, Kearney RE. Intrinsic and reflex contributions to human ankle stiffness: variation with activation level and position. *Exp. Brain Res* 2000;135:423–436. [PubMed: 11156307]
- Moorhouse KM, Granata KP. Trunk stiffness and dynamics during active extension exertions. *J. Biomech.* in press
- Omino K, Hayashi Y. Preparation of dynamic posture and occurrence of low back pain. *Ergonomics* 1992;35:693–707. [PubMed: 1535315]

- Parnianpour M, Campello M, Sheikhzadeh A. The effect of posture on triaxial trunk strength in different directions: its biomechanical consideration with respect to incidence of low-back problems in construction industry. *Intl. J. Ind. Ergon* 1991;8:279–287.
- Punnet L, Fine LJ, Keyserling WM, Herrin GD, Chaffin DB. Back disorders and non-neutral trunk postures of automobile assembly workers. *Scand. J. Work Environ. Health* 1991;17:337–346. [PubMed: 1835131]
- Radebold A, Cholewicki J, Panjabi MM, Patel TC. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. *Spine* 2000;25:947–954. [PubMed: 10767807]
- Rosenbaum D, Hennig EM. The influence of stretching and warm-up exercises on Achilles tendon reflex activity. *J. Sports Sci* 1995;13:481–490. [PubMed: 8850574]
- Shultz SJ, Carcia CR, Perrin DH. Knee joint laxity affects muscle activation patterns in the healthy knee. *J. Electromyogr. Kinesiol* 2004;14:475–483. [PubMed: 15165597]
- Simon, SR. *Orthopaedic Basic Science*. American Academy of Orthopaedic Surgeons; Rosemont, IL: 1994.
- Solomonow M, Zhou B, Harras M, Lu Y, Baratta RV. The ligamento-muscular stabilizing system of the spine. *Spine* 1998;23:2552–2562. [PubMed: 9854754]
- Solomonow M, Zhou B, Baratta RV, Lu Y, Harras M. Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 1. Loss of reflexive muscular stabilization. *Spine* 1999;24:2426–2434. [PubMed: 10626304]
- Solomonow M, Eversull E, He ZB, Baratta RV, Zhu MP. Neuromuscular neutral zones associated with viscoelastic hysteresis during cyclic lumbar flexion. *Spine* 2001;26:E314–E324. [PubMed: 11462097]
- Solomonow M, Baratta RV, Banks A, Freudenberger C, Zhou BH. Flexion-relaxation response to static lumbar flexion in males and females. *Clin. Biomech. (Bristol, Avon.)* 2003a;18:273–279.
- Solomonow M, Baratta RV, Zhou BH, Burger E, Zieske A, Gedalia A. Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. *J. Electromyogr. Kinesiol* 2003b;13:381–396. [PubMed: 12832168]
- Solomonow M, Zhou BH, Baratta RV, Burger E. Biomechanics and electromyography of a cumulative lumbar disorder: response to static flexion. *Clin. Biomech* 2003c;18:890–898.
- Stokes IAF, Gardner-Morse MG, Henry SM, Badger GJ. Decrease in trunk muscular response to perturbation with preactivation of lumbar spinal musculature. *Spine* 2000;25:1957–1964. [PubMed: 10908940]
- Twomey LT, Taylor JR. Flexion creep deformation and hysteresis in the lumbar vertebral column. *Spine* 1982;7:116–122. [PubMed: 7089687]
- Van Dieen JH, Cholewicki J, Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. *Spine* 2003;28:834–841. [PubMed: 12698129]
- Zhang LQ, Rymer WZ, Sliwa JA. System identification of tendon reflex dynamics. *IEEE Trans. Rehabil. Eng* 1999;7:193–203. [PubMed: 10391590]

Fig. 1.
Experimental setup. Subject connected to servomotor for data collection.

Fig. 2.
Force input, erector spinae EMG output and trunk movement in a female subject after the flexion protocol.

Fig. 3. Impulse response function (IRF) for the erector spinae muscle in a female subject after the flexion protocol. The peak reflex gain and rise time are labeled.

Fig. 4. Impulse response function (IRF) relating input force perturbation to trunk movement in a female subject. Kinematic gain, G_K is labeled.

Table 1

Effects of isotonic trunk extension preload

	Men	Women	Average
Reflex gain GR (%/N)			
100N preload	0.987 (0.375) ^a	1.476 (0.846) ^a	1.224 (0.688) ^b
135N preload	1.032 (0.465) ^a	1.467 (0.735) ^a	1.259 (0.640)
170N preload	1.112 (0.418) ^a	1.644 (0.834) ^a	1.370 (0.726) ^b
Average	1.054 (0.420) ^c	1.529 (0.802) ^c	1.285 (0.677)
Reflex latency t _R (ms)			
100N preload	70.70 (17.71)	68.36 (12.07)	69.56 (15.17)
135N preload	68.46 (15.93)	65.45 (7.59)	67.00 (12.59)
170N preload	66.71 (13.85)	63.62 (9.17)	65.21 (11.83)
Average	68.62 (15.84)	65.81 (9.89)	67.26 (13.33)
Kinematic gain G _K (mm/N)			
100N preload	590.4 (90.83) ^d	582.4 (85.70) ^d	598.5 (87.8) ^b
135N preload	557.5 (74.49)	558.6 (73.34) ^d	558.0 (72.9) ^b
170N preload	521.6 (87.88) ^d	511.6 (89.34) ^d	517.2 (87.4) ^b
Average	556.5 (87.93)	559.6 (90.62)	557.9 (88.73)

Mean (standard deviation) of peak reflex gain, latency, and kinematic gain. Results show significant main effects for gender, preload and two-way interactions on reflex gain, GR, and kinematic gain, G_K.

^a Significant effect of gender within preload level $P < 0.05$.

^b Significant main effect for preload $P < 0.05$.

^c Significant main effect for gender $P < 0.05$.

^d Significant effect of preload within gender group $P < 0.05$.

Table 2**Effects of flexion-relaxation (FR)**

	Men	Women	Average
Reflex gain G_R (%/N)			
Before FR	1.070 (0.469)	1.333 (0.635) ^a	1.198 (0.569) ^b
After FR	1.038 (0.369) ^c	1.724 (0.905) ^{ac}	1.371 (0.763) ^b
Average	1.054 (0.420) ^d	1.529 (0.802) ^d	1.285 (0.677)
Reflex latency t_R (ms)			
Before FR	69.39 (15.83)	65.36 (8.28)	67.43 (12.84)
After FR	67.86 (15.97)	66.26 (11.33)	67.08 (13.87)
Average	68.62 (15.84)	65.81 (9.89)	67.26 (13.33)
Kinematic gain G_K (mm/N)			
Before FR	562.1 (95.00)	581.1 (96.19) ^a	570.6 (95.10) ^e
After FR	550.9 (81.47)	538.1 (81.01) ^a	545.2 (80.75) ^e
Average	556.5 (87.93)	559.6 (90.62)	557.9 (88.73)

Mean (standard deviation) of peak reflex gain, latency, and kinematic gain. Interactions between variables of gender, flexion-relaxation effect, and preload are shown. Significances are indicated by superscripts identified in footnotes.

FR = Flexion-relaxation.

^a Significant effect of flexion-relaxation within gender group $P < 0.05$.

^b Main effect for flexion-relaxation approaching significant $P = 0.055$.

^c Significant effect of gender within flexion-relaxation condition $P < 0.05$.

^d Significant main effect for gender $P < 0.01$.

^e Main effect for flexion-relaxation significant $P < 0.01$.

Table 3

Mean (standard deviation) of baseline rectus abdominis activity and pelvic angle

	Men	Women	Average
Rectus Abd EMG (%MVC)			
Before FR	0.0336 (0.0603) ^a	0.0752 (0.1684) ^a	0.0544 (0.1276)
After FR	0.0467 (0.0888) ^a	0.0709 (0.1691) ^a	0.0588 (0.1351)
Average	0.0402 (0.0758)	0.0731 (0.1681)	0.0566 (0.1311)
Pelvic angle (deg)			
Before FR	12.49 (7.65) ^b	13.97 (6.51)	13.10 (7.03) ^c
After FR	14.42 (8.08) ^b	15.51 (5.95)	14.87 (7.09) ^c
Average	13.45 (7.72)	14.74 (6.05)	13.98 (7.01)

FR = Flexion-relaxation.

^a Significant effect of gender within flexion-relaxation condition $P < 0.05$.^b Significant effect of flexion-relaxation within gender group $P < 0.05$.^c Significant main effect for flexion-relaxation $P < 0.05$.