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Influence of Fatigue in Neuromuscular Control of Spinal Stability

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Abstract

Lifting-induced fatigue may influence neuromuscular control of spinal stability. Stability is primarily controlled by muscle recruitment, active muscle stiffness, and reflex response. Fatigue has been observed to affect each of these neuromuscular parameters and may therefore affect spinal stability. A biomechanical model of spinal stability was implemented to evaluate the effects of fatigue on spinal stability. The model included a 6-degree-of-freedom representation of the spine controlled by 12 deformable muscles from which muscle recruitment was determined to simultaneously achieve equilibrium and stability. Fatigue-induced reduction in active muscle stiffness necessitated increased antagonistic cocontraction to maintain stability resulting in increased spinal compression with fatigue. Fatigueinduced reduction in force-generating capacity limited the feasible set of muscle recruitment patterns, thereby restricting the estimated stability of the spine. Electromyographic and trunk kinematics from 21 healthy participants were recorded during sudden-load trials in fatigued and unfatigued states. Empirical data supported the model predictions, demonstrating increased antagonistic cocontraction during fatigued exertions. Results suggest that biomechanical factors including spinal load and stability should be considered when performing ergonomic assessments of fatiguing lifting tasks. Potential applications of this research include a biomechanical tool for the design of administrative ergonomic controls in manual materials handling industries.

INTRODUCTION

The influence of fatigue on the biomechanical etiology of low-back disorders (LBDs) is poorly understood. Musculoskeletal fatigue and endurance contribute to LBD risk, indicating that fatigue may affect spinal load or injury tolerance (Jones, Bovee, Harris, & Cowan, 1993; Macfarlane et al., 1997; Taimela, Kankaanpaa, & Luoto, 1999). The affects of fatigue on spinal load have been previously studied (Marras & Granata, 1997), but there have been no studies of the influence of fatigue on spinal stability or the injury tolerance associated with stability.

Stability is an estimate of musculoskeletal injury tolerance represented by Euler buckling or systems analyses of the neuroanatomic structure (Cholewicki & McGill, 1996; Crisco & Panjabi, 1992; Gardner-Morse, Stokes, & Laible, 1995; Granata & Wilson, 2001). When the spine is stable under a given load, then small neuromuscular or vertebral movement errors are automatically corrected without tissue damage. Conversely, if the spine is unstable, then a small neuromuscular error can be amplified by the biomechanical forces, causing sudden undesired vertebral motion (i.e., spinal column buckling; Crisco & Panjabi). These buckling movements may impose acute strain on intervertebral tissues or stress on the nerve root foramen. To control spinal stability, well-orchestrated neuromuscular control is necessary

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(Cholewicki, Polzhofer, & Radebold, 2000; McGill, 2001; McGill & Cholewicki, 2001). It is well documented that fatigue reduces neuromuscular control of trunk movement (Parnianpour, Nordin, Khanovitz, & Frankel, 1988). Thus fatigue may contribute to LBD risk by limiting injury tolerance associated with stability of the spine.

Injury tolerance may be dramatically influenced by spinal stability. Low-back injuries occur when spinal loads exceed injury tolerance (Herrin, Jaraiedi, & Anderson, 1986). Material failure of the spine may occur at vertebral compression loads in excess of 6400 N, as reflected in national workplace standards (National Institute of Occupational Safety and Health, 1981), and the material tolerance limit is influenced by vibration, repetitive loading, and torsional moment (Brinkman, Biggemann, & Hilweg, 1988; Broberg,1983; Callaghan & McGill, 2001; Shirazi-Adl, Ahmed, & Shrivastava, 1986). However, structural failure of the unsupported spinal column can occur as a result of mechanical instability at compressive loads less than 100 N (Crisco & Panjabi, 1992; Crisco, Panjabi, Yamamoto, & Oxland, 1992; McGill, 2001), causing potential strain injury to the intervertebral tissues and cortical region of the vertebra. Fortunately, the neuromuscular system can augment mechanical stability of the spine, thereby increasing the structural tolerance and allowing it to safely withstand extreme compressive loads (Gardner-Morse et al., 1995). Thus neuromuscular stability control factors may influence injury tolerance of the spine and may influence injury risk.

Neuromuscular control factors contributing to spinal stability are influenced by fatigue. Mechanical stiffness of the musculoskeletal system is a primary component of stability, particularly the stiffness of actively contracting muscles supporting the spine (Gardner-Morse et al., 1995). Fatigue may influence stiffness of actively contracting muscles (Golhoffer, Komi, Miyashita, & Aura, 1987). However, antagonistic coactivation can be recruited to modulate stiffness and augment spinal stability (Cholewicki, Panjabi, & Khachatryan, 1997; Gardner-Morse & Stokes, 1998; Granata & Orishimo, 2001). Thus, in response to fatigue-induced loss of muscle stiffness, it may be necessary to modify coactivation patterns to maintain spinal stability. This outcome has been observed: Modified trunk muscle recruitment and increased coactivation have been noted with fatigue of the trunk musculature (Sparto & Parnianpour, 1998; Sparto et al., 1997). Feedback response and feedback delay also contribute to the stability of dynamic systems (Ogata, 2002). Research has observed modified paraspinal muscle reflex response with fatigue (Magnusson et al., 1996; Wilder et al., 1996), potentially influencing neuromuscular control of movement and spinal stability. Hence there is evidence to suggest that lifting-induced fatigue may influence spinal stability. This fatigue related reduction in stability may contribute to the risk of low-back injury.

The goal of this research was to investigate the potential influence of fatigue on spinal stability. Biomechanical modeling and empirical assessment of lifting biomechanics were performed to test two hypotheses: (a) that reduced force and stiffness capacity in the paraspinal muscles will reduce effective stability of the spine, and (b) that the system will compensate in an attempt to restore trunk stiffness, specifically through recruitment of antagonistic coactivation. The results support our hypotheses and demonstrate that fatigue may impair the neuromuscular control of spinal stability.

METHODS

Model

A biomechanical model was implemented to compute the effects of muscle fatigue on spinal stability. Briefly, a three-dimensional (3-D), two segment model of the spine was developed that included 12 muscle equivalents (Figure 1). The two-segment geometry is unique, allowing independent control of trunk flexion and lumbar lordosis as well as requiring stabilizing support against both global and local buckling behavior as defined by Bergmark (1989). Research

indicates that intervertebral resistance to motion or passive stiffness components contribute little to the stability of the trunk in the functional range of motion and were therefore ignored in the model (Cholewicki et al., 1997). Modeled muscles included the right and left recti abdominis, external obliques, internal obliques, and paraspinal muscles incorporating one- and two segment elements. Muscle origins, insertions, and cross-sectional areas were established from published anatomy (Jorgenson, Marras, Granata, & Wiand, 2001;Marras, Jorgenson, Granata, & Wiand, 2001). Muscle force-generating capacity was modulated by muscle length, as described in published literature (Granata & Marras, 1995a). However, this force-length factor played a negligible role, as all analyses and measurements were performed in a static upright posture only.

The model solved for muscle recruitment patterns that simultaneously satisfied equilibrium and stability. Six degrees of freedom (DOFs) of equilibrium were satisfied by equating the sum of muscle moments with the 3-D static external moments about each spinal segment (Equation 1a); r, f_{m}, and M_{Ext} were the muscle insertion vector, unit-vector of muscle force, and external moment at the vertebrae, respectively.

$$\sum_{m}\left(r\times\hat{f}_{m}\right)F_{m}=M_{\mathrm{Ext}} \tag{1a}$$

$$\mathrm{eig}=\left(\frac{\delta^{2}}{\delta\theta_{i}\,\delta\theta_{j}}V\right)\geq 0 \tag{1b}$$

$$F_{m}<gain\cdot area_{\mathrm{m}} \tag{1c}$$

The set of muscle forces were constrained to satisfy stability, wherein the eigenvalues of the six-DOF Hessian matrix of potential energy, V, must be greater than zero (Equation 1b). The combined equilibrium and stability requirements allow force estimation of up to 12 muscle groups bounded by physiological limits of force-generating capacity (Equation 1c) expressed in terms of muscle cross-sectional area, $area_{\mathrm{m}}$, and peak muscle stress, $gain$ = 50 N/cm^2. Excessive cocontraction can be recruited to increase stability beyond minimum requirements, but it is costly in terms of spinal load and energy expenditure. Therefore, quadratic optimization was performed with an objective function to minimize the sum of muscle stress,

$$\min\sum_{\mathrm{m}=1}^{12}F_{\mathrm{m}}^{\prime}\sigma^{2}F_{\mathrm{m}}, \tag{2}$$

in which F_{m} and σ are the force amplitude and stress-squared matrix of muscles m = 1...12. The analyses were subject to the three constraints of equilibrium, stability, and force capacity (Equations 1a-1c).

Potential energies of the system required for stability calculations were determined from spinal posture and muscle stiffness. (See Cholewicki & McGill, 1996, for a description of potential energy and stability calculation of the musculoskeletal spine.) Muscle stiffness, k, is a well-known physiologic phenomenon describing the deformable behavior of actively contracting muscles (Morgan, 1977). Active muscle stiffness was estimated as a linear function of muscle force, F, and inversely proportional to the muscle equilibrium length, L (Bergmark, 1989),

$$k=qF/L \tag{3}$$

in which q is a unitless constant of proportionality that we shall call the *stiffness gradient.* The nominal stiffness gradient was set at q = 5 for muscles without fatigue, based on results of Gardner-Morse et al. (1995). Spinal load was computed from the vector sum of muscle forces and external load at the lumbar-sacral level. To model the influence of fatigue, we performed

sensitivity analyses to detect the influence of the stiffness gradient, *q*, and the paraspinal force capacity, gain.

Experiment

Twenty-one participants (11 men, 10 women) with no history of low-back pain participated after signing informed consent (Table 1). The protocol required the participants to maintain a static upright posture against a horizontal flexion force of 110 N applied to the trunk. External flexion loads were applied by a weight hung from a Kevlar cord that passed over a pulley and attached to the participant's chest harness (Figure 2). In addition to the preload, flexion perturbations were applied by dropping a 2.27-kg load from heights of 0.5 and 1.0 m, thereby generating sudden flexion moments. Participants were asked to maintain the static upright posture against the extension load and to "not intervene" when perturbed by the sudden load. To restrict lower-body movement during the sudden-load perturbations and to avoid pulling the participants off balance, a waist strap provided restraint while a multiaxis load cell (Bertec PY6-050, Columbus, OH) recorded the restraint forces.

Data from three trials were recorded in each sudden-load condition. Participants completed the sudden-load trials, then participated in a lifting-induced fatigue protocol, and then immediately repeated the sudden-load trials. The fatigue protocol required participants to repeatedly lift a 12.7-kg load from the floor to an upright posture at a rate of 60 lifts/min for a minimum of 2 min or until the rate could not be maintained. A minimum of 2 min rest was provided between trials during baseline (pre-fatigue) measurements, but after the fatigue protocol, the sudden-load trials were performed with no rest between trials. The post-fatigue sudden-load trials could be completed within 1 to 2 min of the fatigue task.

Trunk and spinal motion, kinetics, and trunk muscle activities were recorded during all sudden-load trials. The sudden-load force caused a brief forward flexion movement, which was recorded from surface-mounted electromagnetic sensors (Ascension Technologies, Burlington, VT). Sensors were placed over the participant's spinous processes at T10 and S1, and a third marker was placed on the manubrium. Forces applied to the participant, including components from preload and sudden-load impact forces, were recorded from a load cell in line with the Kevlar tether (Interface SSM-500, Scottsdale, AZ). Ground reaction loads were recorded from a force platform on which the participants stood (Bertec 4060-08, Columbus, OH), and restraining forces were recorded from a load cell attached to the waist belt.

Electromyographic (EMG) signals were collected from bipolar surface electrodes (Medicotest, Rolling Meadows, IL) over four bilateral sets of trunk muscles. Electrodes were placed according to Mirka (1991): for the rectus abdominis, 3 cm lateral and 2 cm superior to the umbilicus; external oblique, 10 cm lateral to the umbilicus with an orientation of 45° to vertical; posterior internal oblique, 8 cm lateral to the midline within the lumbar triangle at a 45° orientation; and erector spinae, 4 cm lateral to the L3 spinous process. Data were band-pass filtered between 15 and 450 Hz, sampled at 1000 Hz.

Analyses were performed to quantify the median power frequency (MPF) of preparatory EMG, mean preparatory integrated EMG (IEMG) amplitude, IEMG response amplitude to sudden load, and trunk displacement response to sudden load. MPF of the preparatory isometric EMG was computed from data recorded during the 200 ms immediately prior to sudden-load impact in order to document neurophysiologic fatigue of the measured muscles (DeLuca, 1997). To achieve IEMG, the recorded data were rectified and integrated using a Hanning weighted convolution filter. The signals were normalized with respect to IEMG achieved from maximum isometric voluntary exertions in trunk flexion, extension, and combined flexion/extension with right and left twist. Mean preparatory IEMG amplitude was determined from the 200-ms pre-impact activity from each trial. Response amplitude was quantified as the IEMG amplitude

difference between preparatory levels and the peak reflex response following the impact (Figure 3). Response latency was recorded as the time from impact until the signal exceeded two standard deviations above the preparatory level (DeLuca, 1997). Trunk flexion angle was computed from the peak angular displacement with respect to the upright posture resulting from the sudden load. Lumbar curvature (lordosis) was established from the difference between the thoracic and sacral marker angles.

Repeated-measures analyses of variance were performed to evaluate the influence of fatigue on the MPF, preparatory IEMG, response amplitude, response latency, lumbar lordosis angle, and trunk deflection following sudden load. Analyses were performed using commercial statistical software (Statistica, 4.5, Statsoft, Inc.) using a significance level of $\alpha < .05$.

RESULTS

Results from the stability model and experimental assessments indicated a potential change in spinal stability with fatigue. A significant decline in the median power frequency of the paraspinal EMG data indicated that these muscles were successfully fatigued by the lifting protocol. No change in the MPF of the oblique or abdominal muscles was noted. Thus in the stability model, only the paraspinal muscles were fatigued. Fatigue was modeled by (a) reducing paraspinal muscle stiffness and (b) limiting the paraspinal muscle force capacity. To model reduced muscle stiffness associated with fatigue, the stiffness-versus-force gradient, *q*, was evaluated at levels ranging from 2 to 10. Based on published estimates (Gardner-Morse et al., 1995), it was assumed that the stiffness gradient for unfatigued muscles was $q = 5$, although others have implemented values of $q = 10$ and higher. For values of $q < 2$ the model could find no feasible set of muscle forces that could maintain spinal stability.

Fatigue-induced reduction in paraspinal muscle stiffness caused increased spinal load when stability was included in the model. Recall that the model output spinal load achieved from the estimated muscle force vectors and external loads. When the model was implemented without the constraint of stability (i.e., ignoring constraint Equation 1b), the spinal load was not affected by the value of the stiffness gradient, *q*. Thus, without consideration of stability criteria, the fatigue-induced loss of muscle stiffness had no effect on equilibrium muscle forces or spinal loads. Conversely, when stability criteria were included in the model, reduced paraspinal muscle stiffness required increased antagonistic cocontraction to maintain spinal stability (i.e., the model predicted increased cocontraction with fatigue). The result of this cocontraction was increased spinal load. According to the model, when stability was included as a constraint, fatigue was associated with a monotonic increase in spinal load (Figure 4). An extreme condition of fatigue-induced loss in muscle stiffness at $q = 2$ resulted in more than twice the spinal load of the nominal unfatigued load at $q = 5$. Thus fatigue-induced changes in modeled stability caused changes in muscle recruitment that affected spinal load.

Fatigue-induced loss in modeled force-generating capacity limited predicted muscle recruitment patterns, thereby causing potential loss of spinal stability. Fatigue-induced loss in force-generating capacity was simulated by evaluating model behavior at paraspinal gain levels between 20 and 50 N/cm^2. Only the paraspinal muscles were fatigued in this manner, with the other modeled muscles remaining at gain = 50 N/cm^2. At high, unfatigued force-generating capacity of gain = 50 N/cm^2, stability could be established for all stiffness gradients $q \geq 2$. At a force-generating capacity of gain = 40 N/cm^2, stability could not be maintained at stiffness gradient $q < 3$. This indicates greater risk of instability failure of the spine if the stiffness of the active muscles should decline with fatigue, as noted earlier. However, for $q \geq 3$, the spinal loads at gain = 40 N/cm^2 were identical to the loads predicted at unfatigued gain = 50 N/cm^2. This trend continued such that nominal unfatigued stiffness gradient $q = 5$ was insufficient to maintain stability at force-generating capacity of gain ≤20 N/cm^2. Thus fatigue-induced

reduction in muscle stiffness caused increased spinal load caused by the necessary recruitment of cocontraction. Simultaneously, fatigue-induced reduction in force-generating capacity limited the availability of cocontraction recruitment, thereby reducing the feasible region of spinal stability.

Empirical data confirmed the model's predictions regarding coactive muscle recruitment. Measured preparatory EMG demonstrated significantly increased activity from both the flexor and paraspinal muscles with fatigue. To compensate for the reduced stiffness of the paraspinal muscles, cocontraction of the flexor musculature was predicted by the model. This was empirically confirmed by noting the measured values of myoelectric antagonistic coactivity-that is, the preparatory EMG from the recti abdominis and external oblique muscles increased significantly during fatigue trials (Table 2), potentially to mitigate the loss in stiffness from paraspinal muscle fatigue. If the increased coactivation sufficiently maintained effective trunk stiffness, then trunk deflection following the suddenload impact must not be influenced by fatigue. Kinematic measurements revealed no significant changes in trunk deflection, preparatory spinal curvature (lordosis), or trunk angle following the fatiguing exertions. Other factors that contribute to stability include the myoelectric response behavior to the sudden perturbation. Results revealed no significant main effects or interactions for EMG response latency or amplitude with fatigue.

DISCUSSION

Lifting-induced fatigue may influence neuromuscular control of spinal stability, thereby affecting injury tolerance of the spinal column. A lay definition of *instability* is the tendency to behave in an unpredictable or erratic manner (Ashton-Miller & Schultz, 1991). From a biomechanical perspective, if the spinal column is unstable, a small neuromuscular error will cause sudden undesired vertebral motion and tissue strain. Fortunately, the neuromuscular system can control spinal stability (McGill & Cholewicki, 2001), thereby increasing structural tolerance and allowing it to safely withstand extreme compressive loads (Gardner-Morse et al., 1995). However, the neuromuscular control of trunk movement is influenced by fatigue (Parnianpour et al., 1988), including changes in active stiffness and force-generating capacity of skeletal muscles (Golhoffer et al., 1987).

Epidemiologic studies suggest that fatigability and endurance contribute to LBD risk (Jones et al., 1993; Macfarlane et al., 1997; Taimela et al., 1999). This indicates that fatigue may affect spinal load or injury tolerance. Previous analyses have reported mixed results regarding the influence of fatigue on spinal load (Marras & Granata, 1997; Potvin & O'Brien, 1998; Sparto & Parnianpour, 1998; Sparto et al., 1997). The goal of the current research was to investigate the potential influence of fatigue on spinal stability.

Results demonstrated that fatigue associated with occupational lifting may compromise spinal stability. Theoretical simulations were supported by empirical data, suggesting that stability was restricted but neuromuscular compensation was recruited to maintain stability through increased coactivation of the antagonistic trunk muscles. Whether this compensation mechanism can be recruited at increased levels of fatigue or following more chronic fatigue protocols (e.g., low-force, long-duration tasks representative of industrial performance) remains to be demonstrated. Modeling results indicated that spinal load may increase monotonically with loss in muscle stiffness (Figure 4). Moreover, the model suggests that it may be more difficult to achieve muscle recruitment patterns necessary to maintain stability as muscle force-generating capacity declines with fatigue. Future studies must examine the biomechanical effects of increasing levels of lifting-induced fatigue, but model results from the current study indicate that spinal stability may be increasingly difficult to maintain with greater levels of fatigue.

A simple feedback control diagram and associated characteristic equation (Figure 5, page 89) can be used to help explain the neuromuscular response to lifting-induced fatigue. To maintain stability, the parenthetical term in the denominator of the control equation must remain greater than zero. This is similar to the modeled constraint that the Hessian matrix of potential energy must be positive definite (Equation 1b). Postures were similar in both fatigued and unfatigued conditions, so one can assume that the gravitational loads ("Grav" in the figure) were unchanged. Therefore, fatigue-induced decrease in paraspinal muscle stiffness, k_{Ext}, may cause reduced stability.

Fortunately, the neuromuscular system can compensate by increasing flexor muscle stiffness, k_{Flex}, and/or increasing reflex response, $Ge^{-s\tau}$, in order to maintain stability. Flexor muscle stiffness is achieved through myoelectric activation of these muscles (Cholewicki, Jurulu, Radebold, Panjabi, & McGill, 1999), indicating that stability may be maintained through antagonistic coactivation of the flexor muscles (Gardner-Morse & Stokes, 1998). Thus the feedback control model and the results from the biomechanical model predict that stability will be maintained through modified trunk muscle recruitment patterns, particularly through increased antagonistic cocontraction. These predicted changes were consistent with the measured preparatory EMG data (Table 2) and agreed with published literature (Granata & Marras, 1996; Potvin & O'Brien,1998; Sparto et al.,1997). Results indicate that fatigue-induced changes in trunk muscle recruitment may be related to spinal stability.

Modified recruitment patterns contribute to spinal load and risk of overload injury. It is noteworthy that fatigue-induced loss in paraspinal muscle stiffness would not influence antagonistic muscle recruitment or spinal load computed from the equilibrium condition alone. Thus, if one neglects spinal stability, it is difficult to explain the increased recruitment of antagonistic activity following fatiguing lifting exertions. When stability constraints are imposed (i.e., when the model is required to search for a biomechanically stable solution), increased cocontraction is predicted that concomitantly increases spinal load, potentially as much as twice the pre-fatigue levels. Increased spinal compression resulting from antagonistic coactivation has been established elsewhere (Granata & Marras, 1995b; Hughes, Bean, & Chaffin, 1995). This is the first study to link fatigue to antagonistic cocontraction and associated spinal load.

Previous research has failed to achieve a consensus regarding the influence of lifting fatigue on spinal load. Some authors have concluded that compression is increased (Marras & Granata, 1997; Potvin & O'Brien, 1998; Sparto et al., 1997), whereas others have reported reduced or unchanged spinal compression (Sparto & Parnianpour, 1998). The variability in the published data illustrates the difficulty in estimating the decline in muscle forces from empirically measured data. Nonetheless, the studies agree that the change in equilibrium and equilibriumrelated spinal load attributable to lifting-induced fatigue is typically small. Our results concur when stability constraints are ignored, demonstrating little change in spinal load from the equilibrium computation. However, when stability constraints are included in the analyses, significant changes in biomechanical loads were noted. These results suggest that spinal stability criteria must be considered in combination with spinal loads when evaluating the biomechanical risk of low-back injury.

Although not explicitly included in the biomechanical model, the system dynamics representation of stability (Figure 5) illustrates that reflex response can contribute to biomechanical stability. Measured values of EMG showed no significant changes in reflex response gain or latency between fatigued states. Conversely, Wilder et al. (1996) and Magnusson et al. (1996) observed delayed paraspinal myoelectric response following sudden-load perturbations in fatigued conditions (e.g., τ in Figure 5). Those studies also noted a nonstatistical trend toward reduced myoelectric response gain (e.g., G in Figure 5).

The differences between those results and data from the current study may be related to the nature and severity of fatigue. The motor control literature is divided regarding neuromuscular reflex response following fatigue. Whereas some have concluded that response latency is delayed following fatigue (Golhoffer et al., 1987; Hagbarth, Bongiovanni, & Nordin, 1995; Hakkinen & Komi, 1983), others have observed no change in latency (Golhoffer et al.; Hortobagyi, Lambert, & Kroll, 1991), depending on the protocol and dependent measure. Most agree that reflex response amplitude is reduced following fatigue (Avela, Kyrolainen, & Komi, 1999; Hagbarth et al.; Hakkinen & Komi; Hortobagyi et al.). This is attributed primarily to fatigue inhibition of alpha motor neurons (Basil-Neto, Cohen, & Hallett, 1996; Garland, 1991; Garland & McComas, 1990; Macefield, Hagbarth, Gorman, Gandevia, & Burke, 1991). If response gain were reduced and response latency delayed, this must further inhibit neuromuscular control of spinal stability. It has been noted by others that reflex latency is compromised in patients with low-back pain (Luoto, Taimela, Alaranta, & Hurri, 1998; Taimela, Osterman, Alaranta, Soukka, & Kujala, 1993). Whether this delayed response contributes to the onset of the disability through reduced spinal stability control or develops in response to the low-back injury remains to be demonstrated.

A decline in physiologic muscle stiffness with fatigue was the primary factor contributing to the change in modeled spinal stability and was the primary cause of predicted increases in antagonistic cocontraction. Results from our laboratory indicate a significant reduction in active muscle stiffness following fatiguing isokinetic exertions (Wilson & Granata, in press).These results are supported by others who have demonstrated a fatigue-related decline in muscle elastic stiffness (Kirsch & Rymer, 1992) and stiffness-related performance criteria (Avela & Komi, 1998a, 1998b; Golhoffer et al., 1987; Horita, Komi, Nicol, & Kyrolainen, 1996; Komi, 2000; Leger & Milner, 2000). However, these conclusions are not unanimous (Moritani, Oddson, & Thorstensson, 1990).

It is necessary to note that the decline in stabilizing potential of the paraspinal muscles, the associated compensatory antagonistic coactivation, and the related increase in spinal load were proportional to the effect size of the muscle stiffness decline with fatigue (Figure 4). Clearly, predicted increases in spinal compression greater than 100% represent an extreme change in active muscle stiffness (i.e., a decline in stiffness gradient from $q = 5$ to $q = 2$). However, fatigue may also be related to greater risk of neuromotor recruitment errors (Parnianpour et al., 1988), thereby contributing to risk of instability injury. Future research must quantify the change in muscle stiffness with fatigue and its relation to musculoskeletal stability.

In conclusion, theoretical analyses indicate that fatigue-related changes in muscle stiffness may reduce the capacity of the paraspinal muscles to stabilize the spine. If fatigue is not severe, then compensatory recruitment of antagonistic cocontraction may restore stability, but this will contribute to increased spinal load and an associated risk of overload injury. Empirical measurements of trunk muscle activity confirmed these findings and agree with the published literature. Thus fatigue-related changes in lifting biomechanics may limit spinal stability and increase spinal load. Neuromuscular compensation was necessary to maintain stability following fatigue, but if the preparatory adaptations cannot be achieved, then the risk of spinal instability and the potential risk of injury may be enhanced when muscles are fatigued. Given the metabolic cost of recruiting antagonistic cocontraction, one must acknowledge that the preparatory compensation may fail to achieve sufficient stabilizing control in fatigued individuals. Our results suggest that industrial ergonomics efforts to control the incidence of low-back injury need to consider fatigue and spinal stability.

Biographies

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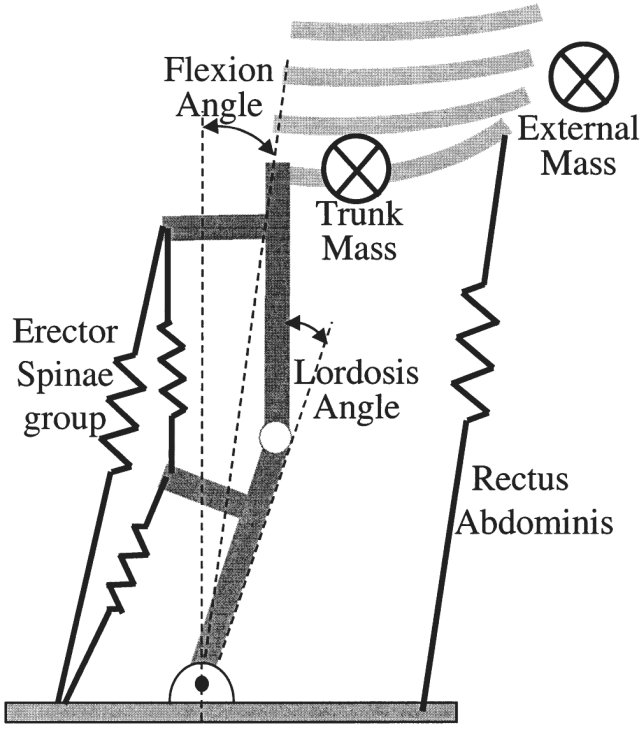

Figure 1.
Schematic representation of the stability model. The spine was represented as a 3-D inverted double pendulum supported by 12 muscles (6 bilateral muscle groups). For clarity, the external oblique and internal oblique muscles have been omitted from the figure but were included in the computational analyses. A more detailed description of the theoretical model is described in Granata and Wilson (2001).

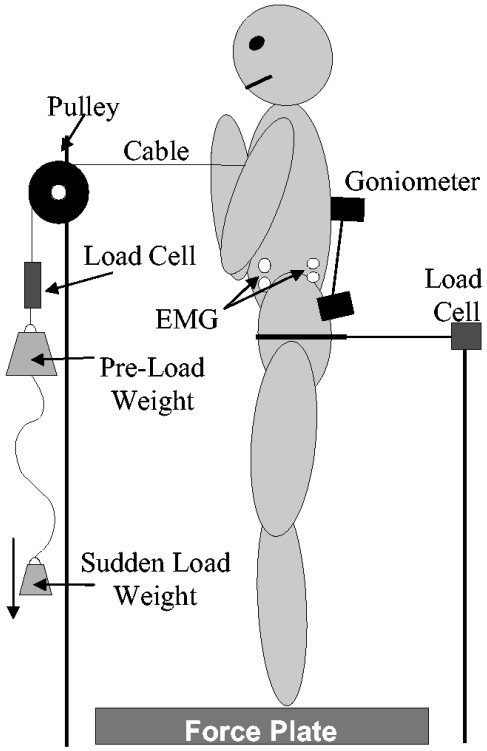

Figure 2.
Experimental setup of sudden load experiment. Preloads to generate external flexion moment and sudden flexion moment perturbations were applied through a cable system. Trunk kinematics, kinetics, and EMG were recorded throughout the disturbance and response. Identical experimental trials were collected before and after extension muscle fatigue.

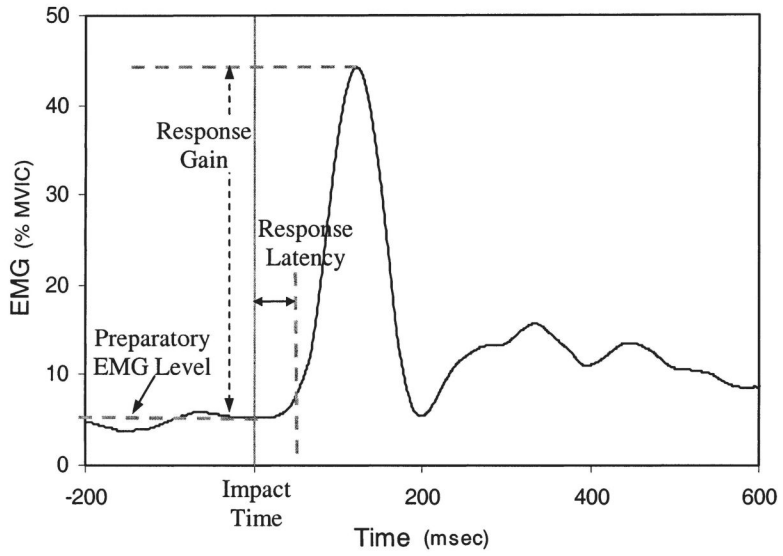

Figure 3.
Typical EMG behavior recorded from the paraspinal muscles. Preparatory mean levels were recorded throughout the 200-ms period prior to the sudden load impact. Response latency and response gain were recorded following the perturbation. Additional analyses were performed to compute the mean power frequency of the isometric preparatory data to establish fatigue level.

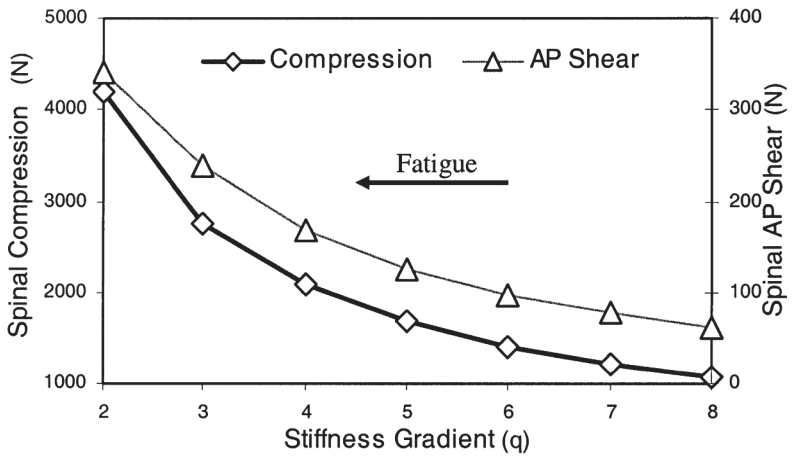

Figure 4.
Reducing the modeled value of muscle stiffness gradient, *q*, requires recruitment of antagonistic cocontraction to achieve stability. Reduced muscle stiffness associated with fatigue causes reduced stability, requiring increased antagonistic cocontraction and resulting in increased spinal compression and anteroposterior (AP) shear load.

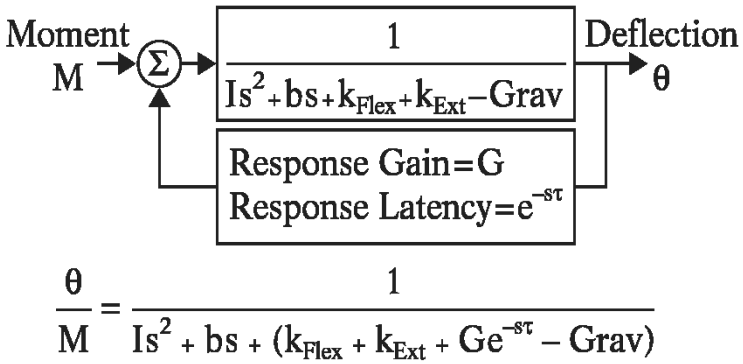

Figure 5.

Control diagram and equation depicting trunk kinematics and muscle reflex. Stability requires that the sum $k_F + k_E + Ge^{-\tau}$ must be greater than gravitational load (Grav). Neither response gain nor response latency changed with fatigue in the measured data. Therefore, if fatigue reduces the stiffness in the trunk extensor muscles k_{Ext}, then increased flexor cocontraction and associated flexor muscle stiffness k_{Flex} must be recruited to maintain stability.

TABLE 1

Participant Demographics

	Men	Women
Number	10	11
Age (years)	20.9 ± 2.3	21.0 ± 3.9
Height (m)*	1.74 ± 0.08	1.62 ± 0.07
Weight (kg)	72.1 ± 10.9	62.9 ± 11.5

* Statistically significant at $p < .05$.

TABLE 2

Measured Kinematic and Trunk Muscle EMG Data From the Experimental Sudden Load Trials

	Prefatigue	Postfatigue
Kinematics (°)		
Lordosis deflection	-8.5 ± 8.36	-7.46 ± 9.14
Trunk deflection	10.21 ± 8.05	10.57 ± 6.63
Preparatory EMG (% MVC)		
Internal obliques	2.8 ± 2.38	3.71 ± 3.55
Recti abdominis*	13.33 ± 4.06	14.23 ± 4.19
External obliques*	6.5 ± 4.19	7.33 ± 4.24
Erector spinae*	4.74 ± 2.84	6.17 ± 4.36
Response amplitude (% MVIC)		
Internal obliques	17.08 ± 8.77	20.15 ± 9.72
Recti abdominis	13.09 ± 6.98	10.81 ± 5.66
External obliques	18.77 ± 6.83	14.71 ± 8.44
Erector spinae	18.75 ± 9.30	19.85 ± 7.79
Response latency (ms)		
Internal obliques	87.1 ± 41.8	78.6 ± 58.1
Recti abdominis	114.8 ± 57.9	130.8 ± 82.8
External obliques	98.70 ± 39.8	90.8 ± 55.7
Erector spinae	126.6 ± 54.5	125.1 ± 66.3
Mean power frequency		
Internal obliques	62.67 ± 17.85	60.93 ± 18.81
Recti abdominis	73.16 ± 25.58	67.91 ± 18.89
External obliques	61.84 ± 17.03	58.56 ± 11.06
Erector spinae*	71.77 ± 15.59	67.45 ± 13.33

*Statistically significant at $p < .05$.