Ergonomics
Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/terg20

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To cite this article: Babak Bazrgari, Brad Hendershot, Khoirul Muslim, Nima Toosizadeh, Maury A. Nussbaum & Michael L. Madigan (2011): Disturbance and recovery of trunk mechanical and neuromuscular behaviours following prolonged trunk flexion: influences of duration and external load on creep-induced effects, Ergonomics, 54:11, 1043-1052

To link to this article: http://dx.doi.org/10.1080/00140139.2011.614357

Ergonomics
Vol. 54, No. 11, November 2011, 1043–1052

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Disturbance and recovery of trunk mechanical and neuromuscular behaviours following prolonged trunk flexion: influences of duration and external load on creep-induced effects

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(*Received 3 June 2011; final version received 8 August 2011*)

Trunk flexion results in adverse mechanical effects on the spine and is associated with a higher incidence of low back pain. To examine the effects of creep deformation on trunk behaviours, participants were exposed to full trunk flexion in several combinations of exposure duration and external load. Trunk mechanical and neuromuscular behaviours were obtained pre- and post-exposure and during recovery using sudden perturbations. Intrinsic trunk stiffness decreased with increasing flexion duration and in the presence of the external load. Recovery of intrinsic stiffness required more time than the exposure duration and was influenced by exposure duration. Reflexive trunk responses increased immediately following exposure but recovered quickly (~ 2.5 min). Alterations in reflexive trunk behaviour following creep deformation exposures may not provide adequate compensation to allow for complete recovery of concurrent reductions in intrinsic stiffness, which may increase the risk of injury due to spinal instability.

Statement of relevance: An increased risk of low back injury may result from flexion-induced disturbances to trunk behaviours. Such effects, however, appear to depend on the type of flexion exposure, and have implications for the design of work involving trunk flexion.

Keywords: low back pain; spine biomechanics; creep deformation; prolonged flexion; stiffness; reflex

1. Introduction

Low back pain (LBP) is the most important work-related musculoskeletal disorder, and continues to have a high prevalence and substantial economic burden (Baldwin 2004, Luo *et al.* 2004, Katz 2006, Dagenais *et al.* 2008, Manchikanti *et al.* 2009). Epidemiological studies have identified several LBP risk factors, in particular prolonged or repetitive torso flexion (Hoogendoorn *et al.* 2000), though some controversy remains regarding causality (Wai *et al.* 2010, Kuijer *et al.* 2011, McGill 2011). Nonetheless, flexed working postures are frequent in mining (Gallagher 2008), construction (Boschman *et al.* 2011), and in agricultural work (Fathallah *et al.* 2008, Fathallah 2010), occupations that all have high LBP incidence rates (BLS 2009). With forward trunk flexion from the upright standing posture, there is a corresponding increase in external moments on the lumbar spine, increasing the requirement for force development in the posterior musculature. Moment arms of these muscles, however, decrease with trunk flexion (Macintosh *et al.* 1993, Jorgensen *et al.* 2003), thereby requiring larger muscle forces for a given external demand and potentially leading to elevated spinal loads in flexed postures (Arjmand *et al.* 2006, Bazrgari *et al.* 2007). Flexed trunk postures may also compromise neuromuscular control of spinal curvature as a consequence of decreased trunk proprioception, leading to spinal instability (Wilson and Granata 2003, Gade and Wilson 2007).

In addition to these adverse mechanical effects of trunk flexion, both active (neuromuscular) and passive (mechanical) trunk tissue behaviours can be influenced if flexed postures occur frequently and/or for prolonged durations. Creep deformation of passive trunk tissues occurs during prolonged flexed postures, manifesting in an increased trunk flexion angle (Twomey and Taylor 1982, McGill and Brown 1992) and an increased activation of the active neuromuscular system (Dickey *et al.* 2003, Solomonow *et al.* 2003b, Shin and Mirka 2007). Alternatively, stress relaxation of passive tissues occurs with sustained flexion and is followed by a substantial decrease in passive spine stiffness (Adams and Dolan 1996) and an angle-dependent decrease in whole-trunk flexural resistance (Hendershot *et al.* 2011). Furthermore, passive stretching of skeletal muscles

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ISSN 0014-0139 print/ISSN 1366-5847 online

http://dx.doi.org/10.1080/00140139.2011.614357
http://www.tandfonline.com

reduces their active force-generating capacity (Fowles *et al*. 2000, Weir *et al*. 2005), diminishes muscle spindle excitability (Avela *et al*. 1999) and may alter the excitability of the ligament-muscle reflex loop (Le *et al*. 2009, Solomonow 2009).

The effects of trunk flexion on passive tissues may compromise spinal stability, which would require increased contributions from the active neuromuscular system. Yet, associated changes in muscle force-generating capacity, muscle spindle excitability, and sensitivity of ligament-muscle reflex loop diminish the efficiency of the neuromuscular system in generating the appropriate responses. Collectively, these changes may adversely affect mechanics of the spinal column and increase injury risk due to excessive spinal loads and/or spinal instability (Panjabi 1992a, 1992b). As such, quantifying the acute changes to active neuromuscular and passive mechanical trunk behaviours and the resultant effects on spine biomechanics (i.e., spinal loads and stability) following prolonged flexed posture are important for better understanding of LBP etiology, and will help improve work design and/or work-rest cycles in occupations involving frequent and/or prolonged flexed postures.

Due to the diversity of occupational exposures, workers may experience different levels of creep deformation and/or stress relaxation, thus requiring separate quantification of the effects of creep deformation and stress relaxation on trunk behaviours. Recently, we have developed a sudden perturbation paradigm to obtain measures related to trunk behaviours, and the effects of flexion angle and duration on active and passive trunk behaviours following a stress relaxation experiment were reported earlier (Hendershot *et al*. 2011). In continuation, the objective of the present work was to investigate the effects of flexion duration and external load on active and passive trunk behaviours following creep deformation. It was hypothesised that (1) the severity of changes in trunk behaviour increases with flexion duration and external load, and (2) recovery is prolonged and contingent on the severity of immediate changes. Moreover, and related to the potential causal role of flexed postures for LBP, we hypothesised that (3) viscoelastic changes in passive trunk tissue following prolonged flexed posture are not adequately compensated by the active neuromuscular system.

2. Methods

2.1. Participants

Twelve young adults participated after completing informed consent procedures approved by the Virginia Tech Institutional Review Board. None had any self-reported history of low-back pain or current medical conditions. Participants included six males with mean (SD) age, stature, and body mass of 23 (3) years, 181.3 (7.9) cm, and 71.3 (7.3) kg, respectively; corresponding values for the six females were 24 (3) years, 166 (6.1) cm, and 60.2 (2.2) kg. A relatively young group of participants was included to avoid potential influences related to age.

2.2. Experimental design and procedures

Creep deformation in the lumbar spine was induced by full trunk flexion in six conditions involving all combinations of three exposure durations (2, 4, and 10 min) and two external loads (none and 29 N). The specific levels of duration and load were intended to represent a range of potential occupational exposures with and without handling a small object (e.g. tool or material). A repeated-measures design was used, in which each participant experienced each condition in a counterbalanced order (one 6 × 6 Latin Square for each gender) and on different days separated by at least 72 h.

Experimental trials imposed full, relaxed flexion of the trunk to induce creep deformation. Participants stood in a rigid metal frame and adjustable straps were used to restrain the pelvis and lower limbs. Subsequently, they slowly ($\sim$3 s) flexed forward from an upright standing and remained in full flexion with minimal trunk muscle activity for the designated duration with their head facing down and their arms relaxed and hanging down (Figure 1). For conditions involving a load, two 14.5 N weights were attached to participants' wrists. Following the flexion exposure, participants returned to and maintained an upright posture with their head facing forward (Figure 1), while still strapped to the frame, for 60 min to assess post-exposure recovery. Recovery measures were obtained 2.5, 5, 10, 20, 30, 40, 50, and 60 min after exposure. The typical delay between the end of a flexion exposure and the first post-exposure measurement was $\sim$15 s.

Participants were instrumented with a 6 DOF inertial measurement unit (IMU: Xsens Technologies XM-B-XB3, Enschede, Netherlands) placed over the T10 vertebral process to measure trunk flexion, and bipolar Ag/AgCl surface electrodes to measure electromyographic (EMG) activity of select bilateral trunk muscles (i.e. erector spinae muscle at the L1 and L3 level, rectus abdominus, and external oblique). Raw EMG signals were pre-amplified ($\times$100) near the collection site, bandpass filtered (10–500 Hz), amplified, and converted to RMS in hardware (Measurement Systems Inc., Ann Arbor, MI, USA). Kinematic data were sampled at 100 Hz and EMG at 1000 Hz.

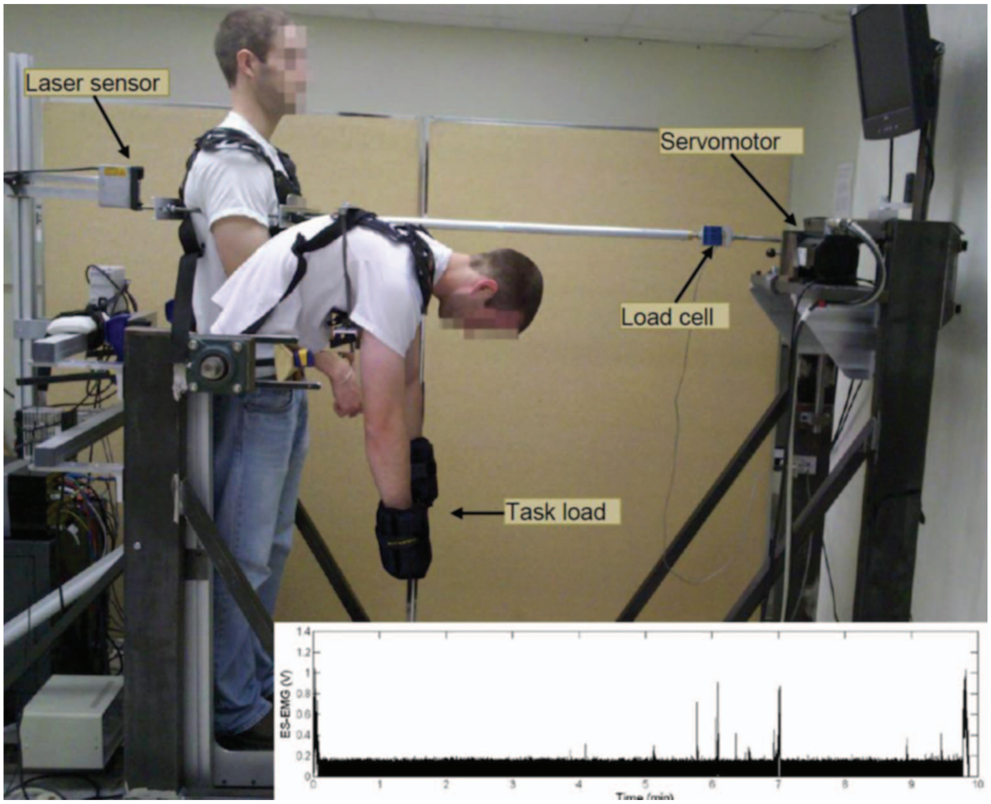

Figure 1. Experimental situation, demonstrating a participant in the sudden perturbation setup superimposed with a picture of the same participant in a flexed posture with external load. A sample of raw erector spinae (L3) EMG, obtained during a 10-min flexion exposure, demonstrates EMG spikes during the creep period. Note that the initial and final EMG activities are related to the initial flexion and final extension phases of exposure.

Pre- and post-exposure measures of trunk behaviours were obtained using a sudden perturbation paradigm identical to that described in our earlier work (Hendershot *et al*. 2011). Briefly, this involved application of a pseudorandom sequence of twelve ±5 mm anterior–posterior perturbations to the trunk (at ~T8) via a servomotor (Kollmorgen, Radford, VA), rigid rod and chest harness (Figure. 1). The total length of perturbation sequences was ~45 s and each perturbation was completed within 40 ms, which is less than typical erector spinae reflex delays. Pseudorandom delays between each perturbation were used to prevent anticipation of perturbation timing by the participants and hence reduce potential confounding from variations in anticipatory muscle activation. Postural displacements were measured with a high-speed, high-accuracy laser displacement sensor (Keyence, Osaka, Japan) and the motor encoder, while applied forces were measured using an in-line load cell (Interface SM2000, Scottsdale, AZ, USA). For ~3 s prior to and during the perturbation sequences, participants maintained a constant submaximal extensor effort (or 'preload'). The target effort was set to 10% of maximum voluntary RMS EMG in the bilateral L3 erector spinae, and which was determined at the beginning of each experimental session. During perturbations, real-time visual feedback of the target effort was provided. Mean (SD) baseline preloads for the submaximal efforts were 62.1 (19.6) N for males and 57 (12.4) N for females.

2.3. *Outcome measures and data analysis*

Creep deformation throughout each exposure period was characterised by changes in trunk flexion measured by the IMU. For each anteriorly directed perturbation, the latent period was determined as the time between perturbation onset and reflexive muscle response (Zhang *et al*. 1999, Granata *et al*. 2004); the former was determined when the absolute value of measured trunk velocity (from laser) exceeded zero, and the latter identified when erector spinae reflex response peaks exceeded two standard deviations above mean activity prior to the perturbations (Hendershot *et al*. 2011). Trunk mechanical behaviours during the latent period (i.e. intrinsic properties) were identified by relating measured trunk kinematics to trunk kinetics (both measured in horizontal direction at T8), and by modelling the trunk as a single degree-of-freedom mass-spring-damper system. An extra mass-spring-damper element was included in the model to account for mechanical properties of connecting elements between the motor and spine, specifically the connecting rod, harness, and soft tissues at the trunk-harness interface (Hendershot *et al.* 2011, Bazrgari *et al.* 2011a, 2011b). In this analysis, trunk damping was forced to zero (see Hendershot *et al.* 2011 for detailed discussion) so that any alterations in intrinsic trunk behaviour could be represented by changes in trunk stiffness; earlier work also suggested that trunk damping may be negligible (Cholewicki *et al.* 2000). Model parameters (apparent mass, stiffness, and damping) were estimated using a least-squares curve fit in MATLAB^{TM} (MathWorks, Natick, MA, USA). To characterise trunk reflexive behaviour, reflex forces were first estimated by subtracting the model-estimated intrinsic force contribution from the total measured trunk response (i.e. trunk reaction force measured by the inline load cell). Magnitude and timing (with respect to perturbation onset) of the maximum reflex force were quantified to represent the overall trunk reflexive behaviour; this analysis was limited to a time window of 150 ms following reflex onset to avoid voluntary responses. The instantaneous reflex force during this same time window was also correlated to time-shifted (equal to reflex delay) trunk velocity to estimate reflex gain (Moorhouse and Granata 2007).

Pre-exposure differences in trunk behaviours between genders were evaluated using unpaired *t*-tests. Post-exposure measures were normalised to pre-exposure values [(post-pre)/pre] and acute effects of flexion duration, load and gender were assessed using mixed-factor analyses of variance (ANOVA). No significant deviations from parametric model assumptions were evident. A repeated-measures MANOVA was used to assess the effects of these same factors over the recovery period (1-h post-exposure); where sphericity violations were found, the Geisser-Greenhouse correction was used. When relevant, post hoc pairwise comparisons were performed using Tukey's honestly significant difference (HSD) test. Data from one trial of a male participant (2 min, no load) and one female participant (4 min, with load) were excluded due to measurement errors. Summary results are presented as means (SD). All analyses were done using JMP^{TM} (Version 8, SAS Institute Inc., Cary, NC), and statistical significance was concluded when $p < 0.05$.

3. Results

3.1. *Pre-exposure*

Estimated apparent trunk mass was larger among males than females (Table 1), and was linearly correlated with whole-body mass ($R^2 = 0.8$). Intrinsic trunk stiffness was similarly higher among males. Muscle reflex delays were comparable between genders, though females demonstrated a significantly larger reflex gain and force magnitude. Initial flexion angles were significantly larger among females than males, with respective values of 80° (19) and 72° (13). Holding the external load increased initial flexion angles by $<1^\circ$ ($t_{(65)} = 0.21$, $p = 0.83$).

3.2. *During exposure*

Creep deformation during flexion exposures across all conditions was 8.1° (4.6). These were not different ($F_{(1,7)} = 0.36$, $p = 0.57$) between females (8.3° (5.9)) and males (7.6° (3.2)). Creep deformation increased with increasing exposure duration ($F_{(2,39)} = 3.87$; $p = 0.03$), with values of 5.9° (3.6), 8.6° (4.5), 9.4° (5.2) following 2, 4, and 10 min of exposure, respectively. External load did not affect creep ($F_{(1,37)} = 0.01$; $p = 0.91$), and the overall difference between loaded and unloaded conditions was $\sim 0.1^\circ$. During flexion exposures, discrete EMG spikes were observed in 76% and 34% of trials among males and

Table 1. Pre-exposure measures of trunk behaviours. Mean (SD) values are shown for each gender, and significant differences between genders are indicated by bolded *p*-values.

Measure	Males	Females	*t*	*p*-value
Apparent mass (kg)	20.3 (3.0)	18.6 (2.3)	$t_{(63.5)} = 2.7$	**0.009**
Intrinsic Stiffness (N/m)	8418 (1201)	5738 (908)	$t_{(63.1)} = 10.4$	**<0.0001**
Reflex delay (ms)	60.1 (3.4)	60.8 (3.4)	$t_{(67)} = -0.9$	0.37
Reflex gain (Ns/m)	1085 (278)	1315 (219)	$t_{(64.5)} = -3.84$	**0.0003**
Max. reflex force (N)	169 (34)	218 (24)	$t_{(61.7)} = -6.93$	**<0.0001**
Timing of Max. reflex force (ms)	158.2 (8.7)	151.8 (6.2)	$t_{(61.3)} = 3.54$	**0.0008**
Initial flexion angle (°)	72 (13)	80 (19)	$t_{(59)} = -2.07$	**0.043**

females, respectively (representative example given in Figure 1).

3.3. *Immediate post-exposure behaviour*

Apparent trunk mass decreased following exposure, but was not affected by duration, load or gender (Table 2). Intrinsic stiffness decreased as exposure duration increased and decreased as the external load increased (Figure 2 and Table 2). Intrinsic stiffness decreased by 4.8% (3.9), 4.7% (4.3), and 9.3% (4.7) following 2, 4, and 10 min of prolonged full-flexed posture and by 7.8% (4.7), and 4.8% (4.5) with and without external load, respectively. Reflex delays were unaffected by flexion exposure; however, reflex gains as well as magnitudes and timing of the maximum reflex forces significantly increased across all conditions, by 79.5 (48.1) Ns/m, 17.7 (6.5) N, and 3.9 (4.7) ms, respectively (Figure 2 and Table 2). Reflex gains were affected by flexion duration, with increases of 3.4% (7.1), 8.3% (7.7) and 10.6% (6) after 2, 4, and 10 min, respectively. Maximum reflex forces increased similarly with flexion duration by 6.8% (8.2), 10.2% (7.8) and 12.1% (5.7) after 2, 4, and 10 min exposures, respectively. Muscle activity (total across all muscles) during the ~3 s prior to perturbations was comparable between all conditions and genders ($p > 0.47$), with respective pre- and post-exposure values of 0.043 (0.009) and 0.044 (0.011) mV.

3.4. *Recovery behaviour*

Apparent trunk mass and reflex delays remained unchanged throughout the recovery period ($p = 0.44$ and $p = 0.76$, respectively). Recovery of intrinsic trunk stiffness was unaffected by external load ($p = 0.49$) or gender ($p = 0.53$), but differed between exposure durations ($p = 0.0006$). Using the criteria of a non-significant difference from pre-exposure values, the time required to recover intrinsic stiffness was directly related to the exposure duration; specifically, 2.5, 20 and 50 min were required following 2, 4 and 10 min of flexion exposure (Figure 3). Recovery of reflex gains, maximum reflex forces, and the timing of maximum reflex in post-exposure perturbation trials were consistent across the three exposure durations ($p = 0.31$, $p = 0.32$ and $p = 0.77$) and two external loads ($p = 0.39$, $p = 0.46$ and $p = 0.74$). Post-exposure changes in the reflex gains and maximum reflex forces were only significant for ~2.5 min following exposure (Figure 4), indicating a fast recovery of reflex behaviour as opposed to intrinsic stiffness. Recovery of reflex behaviours, however, differed significantly between genders. Males exhibited consistently larger reflex gains ($p = 0.022$), larger maximum reflex forces ($p = 0.0007$) and longer times to maximum reflex force ($p = 0.0001$) during recovery from the longest (10 min) flexion exposure (Figure 4). Total muscle activities prior to perturbations remained constant throughout the recovery period and were not affected by condition or gender ($p > 0.32$).

4. Discussion

4.1. *Creep deformation*

Observed creep following 10 min of flexion (mean = 9.4°) was larger than earlier values of ~4° based on lumbar flexion angle (McGill and Brown 1992, Shin and Mirka 2007). Owing to difficulties with attaching an IMU unit on the sacrum in the present study, creep deformation was assessed from trunk rather than lumbar flexion. Though participants' pelvises were restrained in the present study, a portion of the recorded deformation was likely due to creep deformation of lower body parts that were translated into trunk rotation via pelvic rotation. Reports of hamstring soreness by our participants support this idea, as a complete restraining of the pelvis would likely unload the lower limbs and should not lead to such soreness. The difference between our findings and

Table 2. Immediate effects of trunk flexion exposure on trunk behaviours (statistical significance indicated by shaded cells). The 'overall' column indicates paired comparisons (post vs. pre-exposure) across all conditions, and main effects are results from ANOVA (no interaction effects were significant).

		Main effect		
Variable	Overall	Duration	Load	Gender
Apparent mass	$t_{(69)} = -3.79$, $p = 0.0003$	$F_{(2,44)} = 0.3$, $p = 0.75$	$F_{(1,44)} = 0.11$, $p = 0.74$	$F_{(1,10)} = 0.69$, $p = 0.42$
Intrinsic stiffness	$t_{(68)} = -9.92$; $p < 0.0001$	$F_{(2,43)} = 9.9$, $p = 0.0003$	$F_{(1,43)} = 11.5$, $p = 0.001$	$F_{(1,10)} = 3.87$, $p = 0.07$
Reflex delay	$t_{(69)} = 0.98$, $p = 0.33$	$F_{(2,42)} = 1.56$; $p = 0.22$	$F_{(1,42)} = 0.05$; $p = 0.83$	$F_{(1,9)} = 1.2$, $p = 0.31$
Reflex gain	$t_{(69)} = 8.34$; $p < 0.0001$	$F_{(2,43)} = 9.3$, $p = 0.0004$	$F_{(1,43)} = 2.3$, $p = 0.14$	$F_{(1,10)} = 0.7$, $p = 0.41$
Maximum reflex force	$t_{(69)} = 11.2$; $p < 0001$	$F_{(2,43)} = 3.7$, $p = 0.033$	$F_{(1,43)} = 3.1$ $p = 0.086$	$F_{(1,10)} = 1.8$, $p = 0.21$
Timing of maximum reflex force	$t_{(67)} = 4.79$; $p < 0.0001$	$F_{(2,44)} = 0.17$, $p = 0.85$	$F_{(1,44)} = 0.04$, $p = 0.84$	$F_{(1,11)} = 1.7$, $p = 0.21$

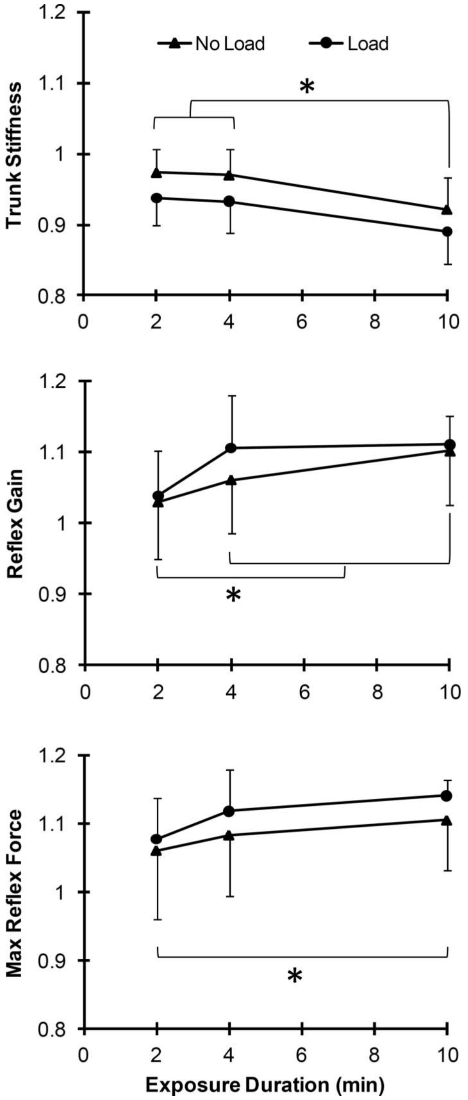

Figure 2. Effects of flexion duration and external load on normalised intrinsic trunk stiffness, reflex gain, and the maximum reflex force. Error bars indicate standard deviations, and * indicates post-hoc groupings with respect to duration.

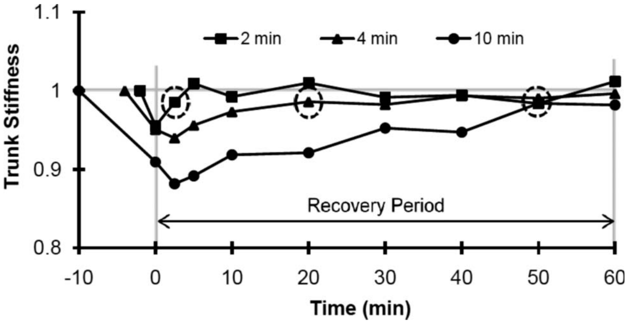

Figure 3. Recovery of normalised intrinsic trunk stiffness following exposure to 2, 4 and 10 min of flexion. Times at which trunk stiffness recovered (i.e. non-significant difference between post- and pre-exposure values) are depicted by the circled data points. Time = 0 indicates the end of the exposure period.

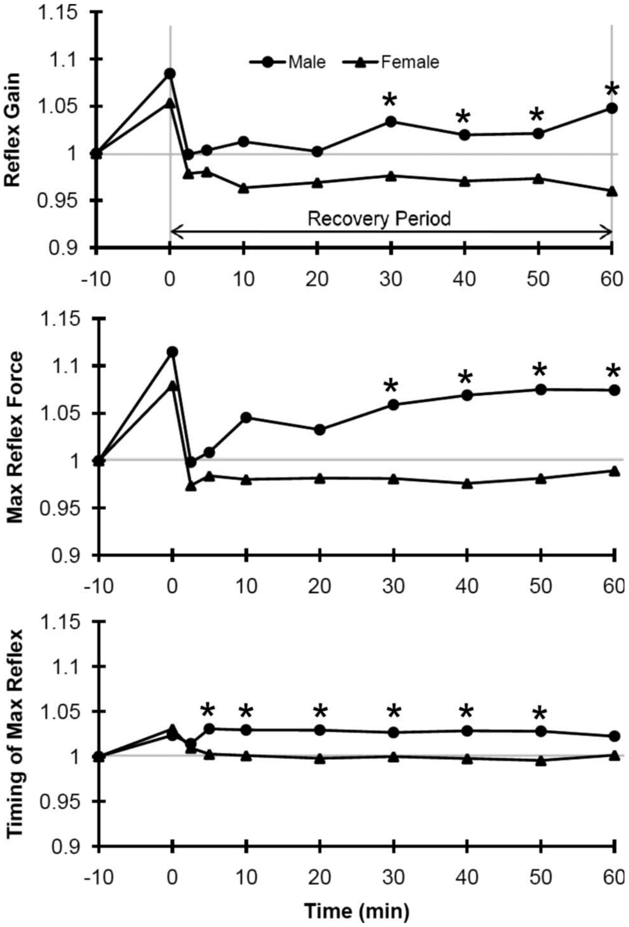

Figure 4. Gender differences in the disturbance and recovery patterns of normalised reflex gain, magnitude, and timing of maximum reflex force following the 10-min flexion exposure. Time = 0 indicates the end of the exposure period and * denotes a significant difference between genders.

those of earlier studies is also consistent with differences in creep deformation in a seated flexed posture based on trunk vs. lumbar flexion angles (Solomonow *et al.* 2003a).

A mean increase of ∼1° in initial trunk flexion angle was found with 29 N of external load, consistent with a ∼1.5–3° increase in trunk flexion relaxation angle reported earlier with ∼100 N external loads (Kippers and Parker 1984, Gupta 2001, Dickey *et al.* 2003). Dickey *et al.* (2003) have also suggested that the lumbar spine may not experience any additional creep with an external load, again consistent with the current results. It appears that the product of trunk mass and some minimum duration (in the present study) is sufficient to bring the trunk viscoelastic tissues to their plateau region of creep deformation. EMG spikes, observed during flexion exposures (cf. Figure 1), have been reported earlier (Solomonow *et al.* 2003a) and have been suggested to result from micro-damage to ligamentous tissue under sustained deviated postures (Solomonow *et al.* 2003a).

4.2. Intrinsic trunk behaviour

Detailed discussions on the methods used to estimate trunk intrinsic properties can be found in our earlier work (Bazrgari *et al.* 2011a, 2011b). Consistent with our earlier studies (Miller *et al.* 2010, Hendershot *et al.* 2011), females demonstrated a smaller intrinsic response (smaller apparent mass and intrinsic stiffness) to sudden perturbations. Since trunk apparent mass was found to be highly correlated with whole-body mass, the prediction of smaller trunk apparent mass for females in the present study can be related to a lower whole-body mass (i.e. 71.3 kg vs. 60.2 kg). The predicted intrinsic stiffness on the other hand is affected by contributions from both laxities of passive tissues in trunk and active stiffness from background muscle activities, both of which have been demonstrated to be significantly smaller in females than males (Rozzi *et al.* 1999, Granata *et al.* 2002a, 2002b). A smaller intrinsic stiffness in females means a less stable trunk than an anthropometrically matched male counterpart. This is consistent with findings of an earlier stability assessment experiment by Granata and Orishimo (2001) wherein it was shown that females demonstrated a significantly higher muscle co-activation to stabilise their trunk, particularly at higher task demands.

Both apparent mass and intrinsic stiffness increase with the level of muscle activity (Cholewicki *et al.* 2000, Gardner-Morse and Stokes 2001, Miller *et al.* 2010). As such, the reduction in apparent mass together with consistent levels of muscle activity found here indicate that exposure-induced decrements in intrinsic stiffness were due to alterations in passive mechanical trunk properties and not changes in background muscle activity (though activity of deeper trunk muscles was not monitored). The decrease in trunk apparent mass following exposure to trunk flexion could be related to changes in the dynamic response from wobbling of trunk soft tissues (Bazrgari *et al.* 2011b). Such a response is in part affected by laxity of soft tissues, which increased following exposure to flexion in the present study. Increased soft tissue laxity results in a smaller inertial response (i.e. less mass is displaced) to a sudden perturbation. The predicted apparent mass in our model is a function of such inertial response, and which is the reason that apparent mass is much less than total trunk mass (i.e. ~50–60% of total body mass), and hence decreased following exposure to trunk flexion.

The observed effects of exposure to flexed postures on intrinsic trunk stiffness supports our hypotheses that both duration and external load increase the severity of changes, and that full recovery requires a duration longer than the initial disturbance time. While creep deformation values in the present study are comparable with earlier investigations, the overall 4–10% decrease in intrinsic stiffness may be an underestimation; measurements were performed in a neutral standing posture, which is associated with the least trunk stiffness (Parkinson *et al.* 2004, Shirazi-Adl 2006). In our earlier study (Hendershot *et al.* 2011), larger relative decreases (i.e. 10–20%) in intrinsic stiffness were found, in large part due to differences in experimental protocol (i.e. stress relaxation vs. creep deformation). Further, a new harness design was used here, which more tightly connected the thorax to the perturbing device and yielded higher estimates of intrinsic stiffness. Otherwise, absolute decreases in intrinsic stiffness in the present study (i.e. 682 (360) N/m) are comparable in magnitude to our earlier results (i.e. 936 (800) N/m).

Recovery of intrinsic stiffness varied directly but non-linearly with exposure duration. Earlier studies have also reported a longer recovery period than the creep exposure time (McGill and Brown 1992, Rogers and Granata 2006, Shin and Mirka 2007), and further suggest a rapid but incomplete recovery of passive stiffness during the initial recovery period. Of note in our results is that trunk intrinsic stiffness continued to decrease during the first few minutes into the recovery period for cases with longer exposure duration. This is in contrast to patterns of intrinsic stiffness recovery following a stress-relaxation protocol, in which recovery was evident immediately after exposure and required a time comparable to exposure duration (Hendershot *et al.* 2011). It is unclear, though, what underlying mechanism is responsible for this difference in recovery from creep vs. load-relaxation exposures.

4.3. Reflexive trunk behaviour

Reflexive trunk behaviours have been investigated using a variety of paradigms (sudden loading vs. unloading, or displacement control vs. force control perturbations), in different trunk postures (upright standing, flexed, supine) and in different loading directions (anteriorly vs. posteriorly). As such, there is a range in reported reflex behaviours (Cresswell *et al.* 1994, Wilder *et al.* 1996, Stokes *et al.* 2000, Granata *et al.* 2004, Rogers and Granata 2006, Moorhouse and Granata 2007, Sanchez-Zuriaga *et al.* 2010, Hendershot *et al.* 2011). Another important factor to consider when comparing the present with earlier results is that three of our reflexive measures (i.e., reflex gain and the timing and magnitude of maximum reflex force) were obtained from mechanical model-estimates of trunk reflexive force. Such measures thus represent a more global measure of trunk reflexive behaviour, differing from EMG-driven estimates of trunk reflexive behaviours. Consistent with one earlier study (Granata *et al.* 2005), we found no effects of flexion exposure on EMG-driven muscle reflex delays. A more recent study (Sanchez-Zuriaga *et al.* 2010), though using a sudden release paradigm in a flexed posture, reported a significant increase in such estimates of reflex delays after creep deformation. The minimum required strain in spinal ligaments to trigger reflex responses increases following ligamentous creep deformation (Le *et al.* 2009). Consistent with this, the timing of maximum reflex force, which was based on the overall trunk reflexive response, did increase following flexion here, suggesting that alterations in reflexive muscle behaviours might have occurred in muscle groups other than those monitored during the experiment. In the Le *et al.* (2009) study, however, spinal ligaments were stretched directly and reflex responses of the adjacent multifidus muscles were recorded, whereas here the whole trunk was flexed and a more global measure of trunk reflexive behaviour was obtained.

Investigations using a feline model suggest a recovery pattern for reflexive muscle behaviour following prolonged static and cyclic flexion-extension that includes an immediate and delayed hyper-excitability, with a period of decreased reflex response in between (Solomonow *et al.* 2003b). Our results confirmed an immediate hyper-excitability and subsequent decrease in the trunk reflexive behaviour in humans. A delayed hyper-excitability period was not evident, though the recovery duration may have been insufficient. While an immediate reflexive hyper-excitability was observed, it did not appear to persist sufficiently to compensate for decreases in intrinsic trunk stiffness (i.e. during the early recovery period), hence supporting our third hypothesis (Figure 5). Following stress-relaxation exposures, this hyper-excitability continued even after complete recovery of trunk intrinsic stiffness (Hendershot *et al.* 2011). Since spinal ligament micro-damage has been suggested as a cause of disturbed neuromuscular behaviour, the differences between our two sets of results may be attributed to differences in experimental protocols, in particular the different level of loadings that can affect the extent of such micro damage. Trunk flexion was achieved in our earlier work by raising the participant's legs while fixing the trunk upright. This resulted in a mean trunk passive resistance of 190 N, which dropped quickly to ~100 N due to stress relaxation, and which was substantially lower than the loading in the current work (i.e. constant trunk weight of ~400 N). As such, the disturbance to and recovery of reflexive behaviour following prolonged trunk flexion appears to be highly dependent on the exposure conditions, in particular the history of trunk loading. In further support, Granata *et al.* (2005), using a similar experimental procedure, reported that muscle reflex gain increased following a single exposure to prolonged flexion, but that gain decreased after four separate exposures with rest between each (Rogers and Granata 2006).

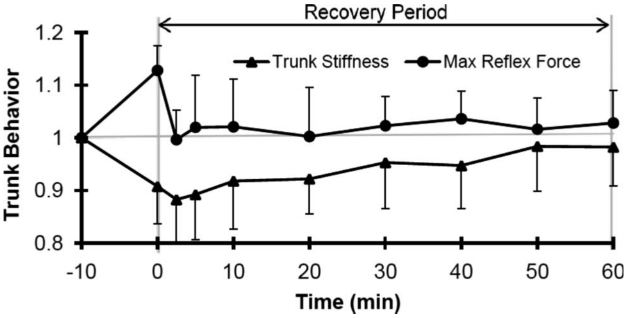

Figure 5. Disturbance and recovery of normalised trunk stiffness and maximum reflex force following exposure to 10 min of flexion. Error bars indicate standard deviations, and time = 0 indicates the end of the exposure period.

Muscle reflexes were significantly larger and faster in females than in males, likely due to higher levels of background muscle activity (Marras *et al.* 2002), and perhaps as compensation for lower contributions from intrinsic stiffness to the control of spinal stability. Females, though, demonstrated a faster recovery of reflex behaviour post-exposure, to a level beyond initial values (Figure 4). This pattern indicates a reduced control of spinal stability following prolonged flexion, and is consistent with the higher incidence rate of LBP in females (Pleis *et al.* 2009).

4.4. *Implications of results*

Occupations involving prolonged or frequent cycles of flexion are associated with higher risks of LBP, particularly when the trunk is flexed >60° for more than 5% of the working time (Hoogendoorn *et al.* 2000). Our findings suggest that this increased risk could be in part due to disturbances in trunk behaviours caused by flexion exposure. Spinal instability has been widely considered as an important cause of LBP (Panjabi 2003). Stability of the spine is provided by force and stiffness contributions from the active trunk subsystem (i.e. voluntary and reflexive muscle responses), passive trunk subsystem (i.e. disc, ligaments and passive muscle), and neuromuscular control subsystem (Panjabi 1992a). Passive trunk stiffness substantially decreased following flexion, and the recovery period exceeded that of the initial exposure. According to the model proposed by Panjabi (1992a), decreases in passive stiffness could be partially compensated by contributions from the active subsystem, through either increases in muscle activation/coactivation or increases in the muscle reflexive response (Moorhouse and Granata 2007, Brown and McGill 2009). However, such expected compensatory responses from the neuromuscular system were either absent (evidenced by constant background muscle activity immediately prior to perturbations) or of insufficient duration (evidenced by the rapid recovery of reflexive responses) to assure system stability. Such a situation can be expected to involve a higher risk of mechanical injury to the spine due to reduced stability. Since a rather long period is required for complete recovery of trunk behaviour, which may not be practically feasible, interventions that change/reduce the exposure type (e.g., exposures that cause stress relaxation rather than creep deformation) or level (extent of flexion) may be more effective than, for example, changing work–rest cycles. Our findings also have potential implications in the design of exercise and rehabilitation programs, such as avoiding exposures that can adversely affect neuromuscular behaviour (e.g., toe-touch stretching). Our participants, however, were university students and were exposed to only one bout of prolonged flexion. As such, future studies are needed to evaluate the effects of exposures among a broader population, longer-term exposures and potential adaptive responses and behaviours.

Acknowledgements

This work was supported by an award (R01OH008504) from the Centres for Disease Control and Prevention (CDC). The contents are solely the responsibility of the authors and do not necessarily represent the official views of the CDC. The authors thank Mr. Waldron and Mr. Vest for their assembly and maintenance of the testing apparatus. The authors dedicate this work to Dr. Kevin P. Granata whose original ideas formed the foundation for the current investigation.

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