A systems-level perspective of the biomechanics of the trunk flexionextension movement: Part II – Fatigued low back conditions

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ABSTRACT

Our companion paper demonstrated the importance of a systems-level perspective on spine biomechanics by showing the effects of lower extremity constraints during simple, trunk flexion-extension motions. This paper explores the impact of trunk muscle fatigue and stress-relaxation of lumbar passive tissues on this systems-level response. Twelve participants performed experimental protocols to achieve lumbar passive tissue stressrelaxation fatigue and lumbar muscle fatigue. Participants performed full range of sagittal-plane trunk flexion-extension under unconstrained stoop movement and pelvic/lower extremity constrained stoop movement. They performed these motions both before and after the fatigue protocols and trunk kinematics and muscle activities in trunk and lower extremity muscles were monitored. Under the condition of passive tissue fatigue, low back muscles and lower extremity muscles revealed significantly increased activation level (21% and 22%, respectively) in the free stoop condition but under the restricted stoop condition, there was no significant effect of the protocol. Under the lumbar muscle fatigue condition, a significant antagonistic and lower extremity activation effect (34% increase in abdominal muscles, 16% increase in lower extremity muscles) was observed in the free stooping condition while these variables were not affected by the protocol under the restricted stooping condition.

Relevance to Industry: Fatigue of the lumbar musculature and passive tissues is prevalent in jobs requiring full trunk flexion postures. Developing accurate biomechanical models of spinal stress in these full stooping postures can help in the development of appropriate interventions to reduce the prevalence of back injuries in these jobs.

Highlights

- Effects of abnormal low back conditions on lifting biomechanics were tested.
- Low back passive tissue fatigue increases lower extremity muscle activity.
- Low back muscle fatigue increases lower extremity muscle activity.
- Interactions between trunk and lower extremity exist under abnormal low back condition.

Key words: Passive tissue elongation; Muscle fatigue; Low back stability; Flexion relaxation phenomenon

1. INTRODUCTION

Abnormal low back conditions such as lumbar muscle fatigue or laxity in ligamentous tissues of the low back have been widely investigated to achieve a better understanding of the mechanism of low back stability (Granata et al., 2004; McGill and Cholewicki, 2001; Rogers and Granata, 2006). Many of these studies have focused on the tissues of the torso and have not considered the potential influence of the structures of the lower extremities; the pelvis often being regarded as a rigid, stable body in most previous models (e.g. Bergmark, 1989; Cholewicki and McGill, 1996; Cholewicki et al., 1998; Granata and Orishimo, 2001; Granata and Rogers, 2007). As discussed in the companion article (Jin and Mirka, 2015), there is evidence in the literature to support a systems-level (i.e., trunk, pelvis and lower extremities) approach for a more comprehensive understanding of trunk stability – particularly at near full flexion postures.

Previous studies have shown that both active and passive tissues in the low back play important roles in providing the necessary restorative moments and spinal stability during trunk flexion and extension exertions (Granata and Rogers, 2007; Granata and Gottipatti, 2008). The flexion-relaxation phenomenon (FRP) has been used as a useful technique to identify the role of the active and passive tissues in achieving spinal stability and biomechanical equilibrium and has been used to study abnormalities of the low back tissues (Colloca and Hinrichs, 2005; Neblett et al., 2003; Watson et al., 1997). The existing literature has a number of studies showing how the condition of the low back tissues (e.g., muscle fatigue and laxity in passive tissues) influence FRP (Descarreaux et al., 2008; Solomonow et al., 2003a; Shin and Mirka, 2007), and demonstrate a reduction of the trunk stiffness and dynamic stability of the torso (Rogers and Granata, 2006;

Granata and Gottipatti, 2008; Granata et al., 2004; Moorhouse and Granata, 2007). Clearly, these studies provide a good theoretical and empirical basis to understand the synergistic interaction between active and passive lumbar tissues around full flexion postures. However, the studies are limited in that they have only focused on the local system (e.g., multifidus muscles) and global system (e.g., lateral erector spinae, rectus abdominis muscles etc.) as proposed by Bergmark (1989), and have not considered the potential influence of the structures of the super global systems (i.e., lower extremities) as proposed by Jin and Mirka (2015).

It is our stance that the local system, the global system and the super global systems are strongly connected, and that the generation of internal trunk extension torque, especially passive moment, for flexion-extension is not only controlled by the local and global system but also influenced by super global system. On this basis, the goal of current study was to understand alternative strategies to supply the necessary moment generation capacity in low back under various abnormal low back conditions. It is hypothesized that the muscle activation pattern of the local, global and super global system will have a complementary interaction to achieve the biomechanical equilibrium between the internal and external moments around the full trunk flexion posture. It is also hypothesized that there will be significant differences in the complementary interactions according to the type of fatigue (muscle or passive tissue) to which the lumbar region is exposed.

2. METHODS

2.1 Participants

Sections 2.1 Participants and 2.2 Apparatus are identical to those of the companion article (Jin and Mirka, 2015).

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2.3. Experimental design

There were two independent variables, POSTURE (two levels: free stooping and restricted stooping) and TIME (two levels: 0 (pre-protocol) and 1 (post-protocol)). The description of the dependent variables is identical to that shown in the companion article (Jin and Mirka, 2015).

2.4. Task and procedure

Upon arrival the experimental procedures were described to the participant and written informed consent was obtained. Participant was fitted with motion and EMG sensors and then performed the MVC exertions. Participants performed two repetitions of the isometric trunk flexion and extension MVC exertions in the lumbar dynamometer while assuming a 20 degree trunk flexion posture. MVC exertions for the gluteus maximus and biceps femoris were performed against manual resistance provided by the experimenter while the participant assumed an upright standing posture (two repetitions for each).

Prior to performing the experimental trials, the participants stood in an open space (no restrictions on pelvis or thighs) in an upright comfortable posture and then bent forward to a full trunk flexion posture. These baseline data defined full range of trunk flexion. The participants were then asked to perform a series of slow, controlled flexion and extension trunk motions consisting of two free stooping trials and two restricted stooping trials. Each of these trials consisted of a 5 second flexion motion (to full flexion), 4 seconds of holding at full flexion and then 5 seconds to extend back to upright posture in time with a metronome sound (one beat per second). The order of the free stooping vs. restricted stooping sequences was randomized across participants and this full-flexion test routine is referred to as TEST.

Upon the completion of the preliminary testing activities, the participants then executed one of three randomly assigned experimental protocols (one week interval between protocols): (1) Protocol A (passive tissue fatigue): alternately perform 25 seconds of full flexion in the seated posture and 5 seconds of upright sitting continuously for 10 minutes; (2) Protocol B (muscular fatigue) alternately perform 25 sec static posture holding at 45 degree trunk flexion in seated posture and 5 seconds of upright sitting continuously for 10 minutes; and (3) Protocol C (combined fatigue) consecutively perform 25 seconds of seated full flexion, 5 seconds of upright sitting, 25 seconds of seated static posture holding at 45 degree trunk flexion. 5 seconds of upright sitting continuously for 10 minutes. These protocols were performed in a seated posture to avoid the confounding effects of lower extremity fatigue. When the 10-min protocol was completed, the TEST routine was performed. The analysis of data from Protocol A and Protocol B are considered in the current paper.

2.5. Data processing

Kinematic Variables. Much of the data processing for the kinematics variables is consistent with that described in the companion article (Jin and Mirka, 2015), but there are some notable variances. The thoracic flexion angle was captured by the difference of the pitch angles between the sensor on xiphoid process and the S1 sensor. The lumbar flexion angle (i.e., lumbar curvature) was captured by the difference of the pitch angles between the T12 sensor and the S1 sensor, representing total movement of the five lumbar spine segments. The EMG-Off variables were expressed as a lumbar flexion angle at which the trunk extensor musculature demonstrated flexion-relaxation phenomenon. The percentage of range of flexion was calculated using the lumbar flexion (LF) angle during flexion-extension. Two calibration data included the LF angle in upright standing measured before each protocol and the LF angle in full flexion measured after the muscle fatigue protocol (Equation 1) (Dolan et al., 1994). The full flexion data after muscle fatigue protocol were employed to provide fair condition to calculate the percentage of flexion in all protocols because less flexion shown in this condition. A preliminary study showed less flexion in the muscle fatigue condition and confirmed that this method guarantees both protocols reach to 100% range of motion; the muscle fatigue condition did not reach to 100% flexion with the calibration data of the LF angle in full flexion captured before experiment.

Percentage Flexion (%) =
$$\frac{[LF - LF_{standing}]}{[LF_{fullflexion} - LF_{standing}]} \times 100$$
 (1)

In the experimental trials the specified range of lumbar flexion angles in which the rectified signals were averaged (herein called the 80-20 range) began during the flexion motion as the participant reached 80% of the full lumbar flexion angle and continued

through the full flexion posture and then ended as the participant passed through that same angle (20% of extension motion) during the returning extension motion. Data processing for EMG data is identical to that of the companion article (Jin and Mirka, 2015).

2.6 Statistical analysis

Statistical analyses were conducted using SAS[®] and Minitab[®]. Assumptions of the ANOVA procedure (homoscedasticity and normality of residuals) were tested before conducting the ANOVA procedures. Appropriate transformations were applied to those data that violated these assumptions until they no longer were in violation (Montgomery, 2001). ANOVA employing a randomized complete block design (blocking on participant) was used to identify significant (p<0.05) main and interaction effects. If a significant interaction between TIME and POSTURE was found, simple effects analysis was conducted to further explore the significance of the main effects.

2. RESULTS

3.1. Protocol A: Passive tissue fatigue

3.1.1. Trunk kinematics

ANOVA showed a significant TIME×POSTURE interaction for both peak lumbar flexion angle and peak thoracic flexion angle (Table 1 and Figure 1), indicating that the restriction of the lower extremities differentially impacted the kinematic response to the elongation of the passive tissues of the lumbar region. Simple effects analysis for peak lumbar flexion angle, sliced by POSTURE, showed a significant difference between TIME 0 and TIME 1 in both free stooping (10% increase) and restricted stooping (4.5% increase). The peak thoracic flexion angle also showed a similar response in free stooping (7.5% increase), but the effect of TIME was not significant.

Considering the EMG-Off lumbar angles for L4 and L3 paraspinals, only the main effects of TIME and POSTURE were significant (Table 1 and Figure 2), showing significantly deeper EMG-Off lumbar angles both after the protocol (TIME 1) and in the restricted posture.

	PROTOCOL A: Pa	ssive Tissue Fatigue	•				
		Dependent varia	bles - Kinematics	Dependent variables - Muscle activity			
Independent variables	Peak lumbar flexion angle	Peak thoracic flexion angle	L3 EMG-off lumbar angle	L4 EMG-off lumbar angle	Agonist	Antagonist	Lower Extremity
Posture	p<0.01	p<0.01	p<0.01	p<0.01	p<0.01	NS	p<0.01
Time	p<0.01	NS*	p<0.01	p<0.01	NS*	NS	NS*
Posture × Time	p<0.05	p<0.01	NS	NS	p<0.01	NS	p<0.05

	PROTOCOL B:	Muscular fatigue					
		Dependent varia	bles - Kinematics	Dependent variables - Muscle activity			
Independent variables	Peak lumbar flexion angle	Peak thoracic flexion angle	L3 EMG-off lumbar angle	L4 EMG-off lumbar angle	Agonist	Antagonist	Lower Extremity
Posture	NS	NS	p<0.01	p<0.01	p<0.01	p<0.01	p<0.01
Time	p<0.01	NS*	p<0.01	p<0.01	NS*	NS*	p<0.01
Posture × Time	NS	p<0.05	NS	NS	p<0.05	p<0.01	NS

Table 1. Summary ANOVA results for Protocol A and Protocol B. (NS – Not Significant; NS* indicates that simple effects analysis revealed that the main effect was not significant.)



Figure 1. Effect of POSTURE and TIME on Peak Lumbar Flexion Angle and Peak Thoracic Flexion Angle in Protocols A and B.



Figure 2. Effect of POSTURE and TIME on L3 EMG-Off Lumbar Angle and L4 EMG-Off Lumbar Angle in Protocols A and B.

3.1.2. Muscle recruitment

Agonist and Lower Extremity (Table 1 and Figure 3). The simple effects analysis for Agonist confirmed a significant effect of TIME in both free and restricted stooping conditions (increase 21% in free and 12% in restricted) but not for POSTURE. Simple effects analysis of Lower Extremity revealed that there was no difference between TIME 0 and TIME 1 in the restricted stooping condition, but showed a significant increase in the free stooping (23% greater). Finally simple effects confirmed significant effect of POSTURE in both TIME 0 and TIME 1 (20% and 42% greater in free stooping, respectively).

The results of the ANOVA showed significant TIME × POSTURE interaction for



Figure 3. Effect of POSTURE and TIME on the NEMG of the Agonist, Antagonist, and Lower Extremity variables collected in the "80-20" range of trunk flexion: Protocol A- Passive Tissue Fatigue.

3.2. Protocol B: Muscle fatigue

3.2.1. Kinematics

The results of the ANOVA showed a significant TIME×POSTURE interaction for peak thoracic flexion angle, but not for peak lumbar angle (Table 1 and Figure 1). Simple effects analysis revealed that neither TIME nor POSTURE were significant main effects for the peak thoracic flexion angle variable but TIME was significant for the peak lumbar flexion angle showing a significant 4% decrease from TIME 0 to TIME 1. Results for the EMG-Off lumbar angle for L4 and L3 paraspinals revealed no significant TIME×POSTURE interactions (Table 1). The main effects of TIME and POSTURE were quite clear with the effect of TIME to make FRP occur earlier for both the L3 and L4 variables (2.6% and 5% earlier, respectively) and with the effect of POSTURE to make FRP occur earlier in the free condition for both L3 and L4 (7.8% and 8.8% earlier, respectively) (Figure 2).

3.2.2. Muscle recruitment

The results of the ANOVA showed significant TIME×POSTURE interaction for Agonist and Antagonist (Table 1 and Figure 4). For both of these muscle groups, simple effects analysis revealed that one of the levels of POSTURE was unaffected by TIME. In the case of the Agonist group the free stooping condition was not affected by muscle fatigue protocol, while the value in the restricted stooping condition increased by 14%. In the case of the Antagonist group the restricted stooping condition was not affected by the fatigue protocol, while the value in the free stooping condition increased by 36%. Considering the Lower Extremity, only the main effects of TIME and POSTURE were significant (Table 1 and Figure 4), showing significantly higher muscle activation levels after the protocol (TIME 1) and in the free posture.



Figure 4. Effect of POSTURE and TIME on the NEMG of the Agonist, Antagonist, and Lower Extremity variables collected in the "80-20" range of trunk flexion: Protocol B – Muscular Fatigue.

3. DISCUSSION

Our companion paper (Jin and Mirka, 2015) provided evidence that supported the need for a more "systems-level" perspective that considers the direct and indirect effects of the lower extremity kinematics and the lower extremity muscle activities on the biomechanics of the lumbar region under "normal" conditions. In the current *in-vivo* study, we explored the impact of the abnormal low back conditions such as trunk muscle fatigue and stress-relaxation of lumbar passive tissues on this systems-level response. These abnormal conditions provided further support for the need for this systems-level perspective.

In general, the passive tissue fatigue protocol generated results that support the results of previous studies. In terms of changes in kinematic variables the results of the current study showed a greater peak lumbar flexion angle and a greater lumbar flexion angle at which the flexion relaxation was initiated as shown in a number of previous studies (Rogers and Granata, 2006; Solomonow et al., 2003a; Shin and Mirka, 2007; Shin et al., 2009). Of particular importance relative to the hypotheses of the current study, were the results that showed a significant interaction between TIME and POSTURE (Figure 1) on peak lumbar flexion angle and peak thoracic flexion angle. This interaction describes a differential response depending on the level of constraint of the lower extremities. The effect of the passive tissue fatigue protocol (moving from Time 0 to Time 1) on these two dependent measures was much greater in the free stooping posture than in the restricted stooping posture. These results suggest a significant role of the lower extremity in free stooping flexion-extension, supplying additional passive moment on low back, and consequently earlier biomechanical equilibrium point between passive tissues and active tissues under the low back passive tissue fatigue condition. Kinematic results not supporting our hypotheses were the results of the EMG-Off angles for the L3 and L4 extensor groups. These results showed the expected deepening of the flexion angle at which flexion-relaxation occurs, but this change from Time 0 to Time 1 was relatively constant across free stooping and restricted stooping postures.

The muscle activation patterns in the passive tissue fatigue protocol revealed a significant interaction between TIME and POSTURE for both the agonist group and the lower extremity group. While all muscle activities increased with the protocol, the increase in the muscle activities in the free stooping condition was more dramatic than

the restricted stooping condition after passive tissue elongation protocol. This response can be attributed to the differences in the stability of the foundation (pelvis) of the two postures. The relative mobility of the pelvis in the free stooping posture would require the recruitment of more low back and lower extremity muscles to create system stability. Previous studies have shown that the foundation (pelvic) stability can be achieved with even slight activation of the erector spinae, the gluteus maximus and the biceps femoris muscles (Vleeming et al., 1989a; Vleeming et al., 1989b; Vleeming et al., 1996; van Wingerden et al., 1993, 2004). Also, Kankaanpää et al. (1998) and Leinonen et al. (2000) suggested importance of achieving the pelvic stabilization in advance of spinal stability after investigating the function of the back and hip extensor in low back patients. This hypothesis is supported by the increase in lower extremity muscle activation levels in the free stooping under the condition and places an emphasis on the role of the lower extremities as an active stabilizer of the pelvis and passive stabilizer on the low back. Collectively, these results that indicate the differentiation between free and restricted stooping support our view that a more systems-level perspective is necessary to fully understand the mechanisms providing equilibrium and stability in the lumbar spine under conditions passive tissue laxity.

Under the low back muscle fatigue condition, the results of the main effects of the fatigue protocol confirmed the trunk kinematics results of previous studies while the interactions showed an interesting response. The results of the current study showed a significant and consistent decrease in the EMG-Off lumbar angle of the trunk extensors after the fatigue protocol (Figure 2) and this response is consistent with that shown by Descarreaux et al. (2008, 2010). Direct comparison with this previous work is challenged

because these authors chose a more global measure (total trunk flexion) while we chose a more local measure (lumbar flexion angle), but the trends in each study are remarkably consistent (-4.5% (current)) vs. (-4.1% (Descarreaux et al., 2010)) in terms of the EMG-Off angles found. The underlying biomechanical/physiological mechanism for reduction in EMG-Off angle is a challenge to describe. Descarreaux et al. (2010) hypothesized that the reduction that they found could be a change in the lumbopelvic rhythm with the lumbar spine accounting for much of the sagittal flexion in the early phases of flexion and the forward pelvic rotation being the primary source of sagittal plane flexion in the latter half of the flexion motion. This model works well when the measure is the EMG-Off angle for total trunk flexion, however, in the current study the measure was a more local measure of lumbar flexion angle which should be independent of the lumbopelvic rhythm and we still observed the same type of response seen in this previous study. One hypothesis is that the activation of the extensor musculature through a prolonged, static exertion could cause a swelling of the muscle through the pooling of blood in the muscle thereby engaging the passive tissues surrounding the muscle (epimysium), creating a passive force/moment through this tissue. Exploration of this hypothesis would be a topic for future research.

The TIME by POSTURE interaction for the muscle activation levels were significant for both the Agonist and Antagonist muscle groups though the trends are a bit different (Figure 4). The antagonist muscle activation did not increase from TIME 0 to TIME 1 in the restricted posture but did so in the free stooping condition indicating a need for the spinal stabilizing effect of antagonist co-contraction. The response of the agonist muscle group followed the opposite trend with the restricted stooping posture

showing a significant increase with fatigue while the free stooping posture was relatively unaffected. Recruitment of additional motor units would be expected because of the decrease in force generation capacity of the fatigued muscles requiring greater activation to keep sufficient spinal stability (Herrmann et al., 2006). However, only the restricted posture revealed this expected significant increase. This limited response in the free stooping posture may be caused by less lumbar flexion angle (i.e., decreased lumbar curvature) and earlier FRP (i.e., FRP in less flexed posture) after the muscle fatigue protocol causing an earlier transition from the active mechanism to the passive mechanism in the free stooping technique.

The alteration of the load-sharing mechanism between active and passive tissues in the low back after the muscle fatigue protocol may be a signal of trunk instability that is caused by inability in low back muscles (Granata et al., 2004; Granata and Gottipatti, 2008). Regarding the role of the antagonist, prior studies pointed out a unique role of the antagonistic exertion under the trunk instability condition. They proposed and showed that the muscles do not contribute to generating any driving force in the movement, but the additional excessive activation stiffens the spinal column and enhances spinal stability to prevent buckling of the spine under the muscle fatigue in low back (Bergmark, 1989, Cholewicki and McGill, 1996, Parnianpout et al., 1988). In addition, the biomechanical models of the spine suggested an increase of trunk stiffness by the recruitment of antagonistic coactivation (Granata and Orishimo, 2001; Crisco and Panjabi, 1990; Gardner-Morse and Stokes, 1998; Granata and Marras, 2000). The result of current study also revealed a significant increase in the antagonistic coactivation after the muscle fatigue protocol in free stooping to enhance trunk stiffness. The earlier transition

into the passive mechanism and limited moment generation capacity of the fatigued low back muscles after the protocol may reduce the spinal stability and hence the antagonist activity was increased to keep the stability around the full flexion. Recall that the muscle fatigue protocol in the free stooping showed earlier transition to the passive mechanism among all conditions (about 2.5° earlier than the restricted stooping). It is possible that the increased passive elastic tension of the fatigued low back muscles can account for the external moment in a similar fashion to the tensed rope, but cannot provide enough stability in the spinal column because of totally different origination and insertion of the muscles than the ligaments. Consequently, the low back system may require additional spinal stabilization mechanism such as coactivation in antagonist.

The lower extremity also showed a significant increase in the free stooping posture after the protocol. As already discussed in the passive tissue fatigue condition, the results suggest the increased role of the lower extremity as a pelvic stabilizer under the condition of the spinal instability (i.e., after the muscle fatigue protocol) for providing a stable foundation and a passive torque. Based on these, the global and super global systems including the Antagonist and Lower Extremity play a key role in the trunk system stability under abnormal conditions such as low back muscle fatigue.

The extension of the Reeves et al. (2007) ball-on-curved-surface example discussed in our companion paper (Jin and Mirka, 2015) also could be employed to explain the difference between two stooping techniques in Agonist, Antagonist and Lower Extremity muscle activation patterns. For an example, the stable pelvis in the restricted posture may not need to have significant recruitment of the passive mechanism by the activation of Antagonist and Lower Extremity after the fatigue protocols; a significant recruitment of

Agonist, especially local system, could be enough to hold the destabilized spinal column (i.e., disturbed ball) with the stable basement. On the other hand, when the bottom of the bowl is required to be stabilized in advance to achieve the ball stability such as the free stooping condition, the low back system could achieve the pelvic and spinal stability by recruiting the Antagonist and Lower Extremity. Our companion paper (Jin and Mirka, 2015) supports a strategy to supply the necessary moment generation capacity in low back under the normal condition by employing the lower extremity, and the current study confirmed that the low back systems including lower extremity have a complementary interaction under the abnormal condition to achieve the biomechanical equilibrium between the internal and external moments around the full flexion posture.

4. CONCLUSION

Our *in-vivo* experiments revealed the importance of considering a "super-global system" model of the lumbar spine by documenting significant differences in kinematic and muscle activation responses as a result of lower extremity constraint under the two abnormal low back conditions. Collectively, the results support the analogy of 'the ball in a stable bowl' model describing the importance of considering the super global system as a part of the trunk flexion-extension system.

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