Altered muscle recruitment during extension from trunk flexion in low back pain developers

Erika Nelson-Wong, Brendan Alex, David Csepe, Denver Lancaster, Jack P. Callaghan

1. Introduction

Eighty percent of all individuals will suffer from low back pain (LBP) at some point in their life-time (Wong and Lee, 2004). In 85% of LBP cases, there is no clear injury mechanism that can be identified as the source for the disorder, and these cases are typically referred to as ‘non-specific’ (Waddell, 2004). Multiple case–control studies have discovered neuromuscular differences between individuals with and without non-specific LBP (Brumagne et al., 2008; Esola et al., 1996; van Dieen et al., 2003). One functional movement that has been commonly investigated in case–control studies is trunk flexion and extension performed while standing. Altered relaxation responses of the extensor musculature have been observed in people with LBP (Alschuler et al., 2009) as well as differences in muscle recruitment strategy and trunk/hip kinematics during performance of the movement (Esola et al., 1996; Wong and Lee, 2004). The typical activation order for extensor muscle recruitment during the extension phase of a standing flexion task occurs in a caudal to cephalic sequence in healthy control subjects (McGorry et al., 2001). McChure et al. (1997) described the typical movement pattern during extension from trunk flexion as being dominated by hip movement during the first 75% of the motion in healthy individuals. A movement pattern where a greater percentage of the extension motion originates from the lumbar spine than from the hip is considered a ‘spine dominant’ strategy. Previous investigations have found that subjects with LBP demonstrated a spine dominant strategy compared with healthy controls (Esola et al., 1996) and premature activation of lumbar paraspinals during the extension phase of the movement (Wong and Lee, 2004).

While it has been accepted that differences exist between people with and without LBP, less well established is whether neuromuscular differences are present prior to the LBP condition and are perhaps contributory factors to LBP development. A functional standing model has been successfully used to induce transient LBP in previously asymptomatic individuals (Gallagher et al., in press; Gregory and Callaghan, 2008; Marshall et al., 2011; Nelson-Wong and Callaghan, 2010b). The model allows for characterization of differences between LBP developers (PD) and non-developers (NPD), prior to pain development, as only a percentage (40–60%) of people exposed to this protocol are found to

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develop LBP (Gregory and Callaghan, 2008; Marshall et al., 2011; Nelson-Wong and Callaghan, 2010b). Using this model, alterations in neuromuscular control, particularly in the frontal plane of movement, have been identified in previously asymptomatic people who are classified as pain developers when exposed to prolonged standing (Nelson-Wong and Callaghan, 2010b; Nelson-Wong et al., 2008). In the sagittal plane, it was found that PD exhibited an increased relaxation response of the gluteal muscles during standing trunk flexion compared to NPD, although no differences were found in the lumbar extensor musculature (Nelson-Wong and Callaghan, 2010b). While case control differences have been described in the literature (Esola et al., 1996; Wong and Lee, 2004), timing and sequencing of extensor muscle activation during extension from trunk flexion were not characterized prior to LBP development in the aforementioned prolonged standing studies. Additionally, although the previous studies found no gender differences in pain reporting rates, gender differences were identified in postural control, neuromuscular strategies, and response to interventions (Gallagher et al., in press; Nelson-Wong and Callaghan, 2010a, 2010b, 2010c). Movement strategy differences related to gender have also been identified in literature investigating knee mechanics and injury, where gender does appear to be a factor in injury rates (Beaulieu et al., 2008; Joseph et al., 2011).

The purpose of the current study was to examine differences in neuromuscular strategies, assessed using recruitment timing between muscle pairs, during the extension phase following trunk flexion between individuals classified as PD and NPD with the induced LBP model. It was hypothesized that the PD and NPD groups would demonstrate differences in neuromuscular strategies during extension from trunk flexion, and that PD would demonstrate a more spine dominant strategy, similar to the pattern that has been observed in patients with LBP (Wong and Lee, 2004). Although not a primary aim of this study, gender differences during the movement were also of interest, with the expectation that males and females would demonstrate different muscle recruitment strategies for the extension phase of trunk flexion.

2. Methods

Experimental data were collected at the University of Waterloo and IRB approval was received through the University of Waterloo Office for Research Ethics. Data analysis for the current study took place at Regis University, and was determined to be exempt by the Regis University IRB.

Forty-three participants (age range 18–33 years old, 22 male) from the University of Waterloo and surrounding community volunteered for this study (Table 1). Participants were excluded from participation if they had any lifetime history of LBP that was severe enough to seek medical care or that required greater than 3 days off from work or school; prior hip or spine surgery; were unable to stand for greater than 4 hours; inability to complete questionnaires; or had been employed within 12 months in an occupation requiring prolonged standing. Informed consent was obtained prior to participation. Detailed methods have been published previously, and readers are encouraged to refer to these earlier publications for complete data collection parameters and signal processing details (Nelson-Wong and Callaghan, 2010b; Nelson-Wong et al., 2010). In brief, participants completed several questionnaires to assess attitudes towards pain, injury and disability to determine if these were factors in their response to the pain inducing protocol. There were no group differences in the questionnaire responses and these findings have been published elsewhere (Nelson-Wong and Callaghan, 2010b). Participants also completed a physical activity questionnaire, Minnesota Leisure Time Physical Activity Questionnaire (Folsom et al., 1986) for the 30 days prior to entrance into the study to determine equivalence in physical activity status at baseline.

Continuous surface electromyography (EMG) data (AMT-8, Bortec, Calgary, Canada) were collected from bilateral thoracic erector spinae (TES), lumbar erector spinae (LES) and gluteus maximus (GMax) muscles (sampling frequency of 2048 Hz). Kinematic data were simultaneously collected (sampling frequency of 32 Hz) with an OptoTrak Certus system (Northern Digital Instruments, Waterloo, Canada) to create an 8-segment (trunk, pelvis, bilateral thighs, shanks, and feet) rigid link model using Visual3D software (C-Motion, Inc., Germantown, MD) for calculation of relative joint angles, trunk velocity, and to track segmental movement. EMG data had high frequency and 60 Hz electrical noise components removed (bandpass filter 10–500 Hz, bandstop filter 59–61 Hz) were full wave rectified and low pass filtered (zero phase lag, Butterworth filter with effective cutoff frequency of 2.5 Hz) and were normalized to maximal voluntary contractions to be expressed as %MVC (Nelson-Wong and Callaghan, 2010b).

Participants were asked to perform a series of self-paced movements, including standing trunk flexion, prior to entering into the 2-hour standing protocol. During the 2-hrs of standing, participants were asked to rate their LBP on a 100 mm Visual Analog Scale (VAS) with end point anchors of 0 mm being no pain and 100 mm being worst pain ever experienced, every 15 min. All participants reported 0 mm on the VAS upon entrance into the study. A threshold of ≥10 mm increase in LBP was used to categorize participants as PD or NPD (Nelson-Wong and Callaghan, 2010b).

The contributions to total trunk flexion range of motion from the hip (thigh relative to pelvis segment) and lumbar spine (trunk relative to pelvis segment) at terminal flexion were calculated by subtracting the neutral standing angle from the maximum angle at terminal flexion. The ratio of lumbar motion to hip motion was then calculated for terminal flexion. The extension phase of each trunk flexion trial was isolated using the kinematic data (trunk angle calculated as thorax relative to pelvis) to determine the instant of terminal flexion, and the frame numbers extracted to determine the appropriate window for the EMG data. Angular velocity of the trunk segment was also calculated for the extension phase of each trunk flexion trial. Cross-correlation was then used to determine relative phase relationships between activation of muscle pairs and muscle activation relative to pelvis position during extension from trunk flexion.

Using cross-correlation (Eq. (1)), spatial and temporal similarity can be determined between two time-varying signals, x(t) and y(t). In brief, one signal, x(t), is held stationary while the second signal, y(t), is time-shifted incrementally forwards and backwards over the total length of the record N, with a spatial correlation, Rxy, calculated at each time shift, τ, to create a cross-correlation function, Rxy(τ). The value for τ at the point of maximum spatial correlation can be considered to be the phase lag between the two signals (Nelson-Wong et al., 2009).

$$R_{xy}(\tau) = \frac{1}{N} \sum_{i=1}^{N} (x_i - \bar{x})(y_i,\tau - \bar{y})$$

$$\bar{y} = \frac{1}{N} \sum_{i=1}^{N} y_i, ~ \bar{x} = \frac{1}{N} \sum_{i=1}^{N} x_i$$

Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>mean (SE)</th>
<th>Independent t-test P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) PD</td>
<td>26</td>
<td>22.50 (0.61)</td>
<td>0.56</td>
</tr>
<tr>
<td>PD</td>
<td>17</td>
<td>23.12 (0.91)</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²) PD</td>
<td>26</td>
<td>23.68 (0.64)</td>
<td>0.84</td>
</tr>
<tr>
<td>NPD</td>
<td>17</td>
<td>23.88 (0.80)</td>
<td></td>
</tr>
<tr>
<td>Activity Level</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MPAQ Score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td>26</td>
<td>14368 (7733)</td>
<td>0.35</td>
</tr>
<tr>
<td>NPD</td>
<td>17</td>
<td>16762 (8928)</td>
<td></td>
</tr>
</tbody>
</table>

MPAQ = Minnesota Leisure Time Physical Activity Questionnaire.
For this study, the following cross-correlations were performed to provide relative sequencing/timing information between two signals, using custom software written in Matlab version 2011b (MathWorks, Natick, MA) for both right and left sides: TES-LES (right and left), TES-GMax (right and left), LES-GMax (right and left), LES-Pelvis position (right and left), and GMax-Pelvis position (right and left). Phase lags, \( \tau \), at maximum \( R_p \) for each of these 10 pairs were extracted and recorded. As a data reduction measure, paired t-tests were used to determine symmetry between left and right phase lags, with \( p \geq .05 \) (no significant differences between sides) indicating symmetrical muscle activation. When left/right symmetry was determined, an average value was taken for left/right phase lags, leaving 5 variables for the analysis.

Independent t-tests were conducted on age, Body Mass Index (BMI), and physical activity data to ensure the PD/NPD groups were equivalent in those factors. To determine differences in neuromuscular strategy, lumbar/hip ratios at terminal flexion, trunk velocities and phase lags were entered into two-way ANOVAs with between factors of PD/NPD group and gender with a significance criterion of \( \alpha \leq 0.05 \). SPSS Version 19.0 (IBM, Armonk, NY) was used for all statistical analysis.

### 3. Results

Seventeen of the 43 participants (40%) were classified as PD with an average increase in LBP VAS of 22.7 (±2.91) mm versus 1.37 (±0.45) mm for NPD (Fig. 1). Seven of the 17 PD and 15 of the 26 NPD were male. PD and NPD participants were found to be equivalent on age, BMI and physical activity level at baseline (Table 1). Because left/right comparisons yielded no significant differences between sides for trunk velocities during the extension phase of return to stand from flexion. There were also no significant main effects of PD/NPD group or gender, and no interactions found for trunk velocities during this sagittal plane movement, indicating the muscle activation sequencing and pelvis movement. There was no significant PD/NPD group by gender interaction for the muscle activation timing comparisons.

A significant main effect of PD/NPD group (\( F_{1,39} = 5.22, \ p = .03 \)) was found for the phase lag between LES and GMmax muscle pairs. PD activated LES 0.039 (±0.086) s prior to GMmax while NPD activated GMmax 0.196 (±0.071) s prior to LES (Fig. 2). The other muscle pairs (TES-Gmax, and TES-LES) showed a similar directionality, however these comparisons failed to reach statistical significance. Table 2 displays summary data for phase lags between muscle pairs and range of motion contributions by gender.

### 4. Discussion

Findings from this study show a cephalic to caudal muscle activation strategy during extension from trunk flexion in individuals who developed pain during standing compared with individuals who did not, supporting the primary hypothesis that pain developers would utilize a spine dominant strategy during trunk extension. Gender differences were also found in lumbar/hip ratios and muscle activation sequencing during this sagittal plane movement, with females exhibiting a hip initiation strategy compared with males, although these differences were independent of PD/NPD group.

The NPD group in this study demonstrated a typical activation order of caudal to cephalic sequence, similar to what has been described previously in healthy control subjects (McGorry et al., 2001). In this study, individuals classified as PD demonstrated an atypical ‘top-down’ recruitment strategy with lumbar extensors being activated prior to gluteal musculature compared to their NPD counterparts. This is of special interest as these subjects were not a clinical LBP sample, but did respond to an induced pain model with development of LBP. Furthermore, the altered muscle recruitment strategy was present prior to exposure to the pain inducing protocol, and subjects were painfree at the time they performed the standing trunk flexion trial. Movements were performed at a self-selected speed, however there were no significant differences between groups or genders in speed of movement, indicating the muscle activation differences were independent of movement pacing. Implications from these findings are that neuromuscular differences exist a priori between individuals who do and do not exhibit a LBP response to standing. These results support similar findings, published elsewhere, from these data that found altered muscle activation profiles during standing as well as an increased relaxation response of the gluteal muscles during terminal flexion in PD (Gallagher et al., in press; Nelson-Wong and Callaghan, 2010b). The findings of delayed gluteal muscle activation during extension from trunk flexion, as well as increased relaxation of gluteal musculature during terminal flexion, in PD are consistent with previous studies that have proposed gluteal muscle inhibition exists in people with LBP (Bullock-Saxton et al., 1993).
Published case-control studies have investigated differences in recruitment strategy during standing trunk flexion and extension between LBP cases and healthy controls. Findings from this study on asymptomatic individuals are similar to reported reversals of activation order during extension (Wong and Lee, 2004), and decreased gluteus maximus activation (Leinonen et al., 2000) in people with LBP. Leinonen et al. (2000) also found that patients with LBP responded to an exercise based intervention by demonstrating earlier activation of gluteus maximus during extension from trunk flexion, further suggesting that this is a normalized recruitment strategy. It appears that individuals with no prior history of LBP, who responded to prolonged standing with reports of LBP, demonstrated similarities in their biomechanical profiles to patients with LBP. This finding suggests that these altered strategies may be present before the development of the LBP disorder, and could be considered as a factor that might prove useful in early identification of people at risk for future LBP.

Abnormal sequencing of trunk musculature may create alterations in biomechanical loading through the lumbar spine that may not otherwise have occurred with a caudal to cephalic activation sequence (Colloca and Hinrichs, 2005). Early activation of lumbar paraspinals (or delayed activation of gluteal muscles), during extension from trunk flexion or a reversal of the normal pattern of sequencing may exacerbate maladaptive loading patterns in the lumbar spine and could potentially contribute to back pain.

It is interesting that there were gender differences detected in TES-GMax timing during extension from trunk flexion. This could be due to anthropometric gender differences such as males carrying a greater percentage of their total body mass in their trunk leading to earlier activation of thoracic musculature. Hoffman et al. (2012) recently described gender differences in relative hip and lumbar spine contribution to standing trunk flexion range of motion, with males having a greater percentage of the total motion originating from the lumbar spine and females having a greater contribution from the hip. Findings from the current study are similar, and this could explain the gender differences in muscle activation timing that were observed. Females had a larger percentage of their total standing flexion arising from the hip, and also activated the hip musculature first during the extension back into standing. There were no gender differences in velocity of movement or in total range of motion, therefore the females in this study would have spent relatively more time moving at the hip while males would have spent relatively more time moving at the trunk, which is consistent with the muscle activation timing findings.

There are some limitations to this study. The participants in this study were relatively young (18–33 years old) and therefore may not be representative of the clinical LBP population. Participants were allowed to move at a self-selected pace, which may have been a confounding factor, although there were no significant differences found in velocity of movement. The trunk was treated as a single rigid segment, which did not allow for a realistic representation of the lumbopelvic kinematics. The relationship between pain development during a bout of prolonged standing and future development of clinical LBP has not yet been determined and this is an area of ongoing research.

5. Conclusion

In conclusion, previously asymptomatic individuals who developed LBP during exposure to a 2-hour standing protocol displayed altered neuromuscular strategies compared to individuals who did not develop LBP. Specifically, lumbar paraspinals were activated earlier than gluteus maximus in pain developers during extension from trunk flexion, which a reversal of the expected and typical activation sequence present in trunk extension from full standing flexion. Identification of predisposing factors for LBP development has the potential to aid health care professionals in early intervention and possibly prevention of LBP.

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