RECRUITMENT OF MUSCULATURE DURING EXTENSION FROM FULL TRUNK FLEXION IS ALTERED IN PEOPLE WHO DEVELOP LOW BACK PAIN DURING PROLONGED STANDING

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INTRODUCTION

Altered muscle activation patterns during lumbar flexion and extension movements have been previously reported in people with clinical low back pain (LBP). A period of electromyographic silence of the lumbar extensors at peak lumbar flexion has been observed in healthy subjects and has been termed the flexion relaxation response. Impairment of this response, shown as a decrease in relaxation has been observed in patients with LBP [1,2]. McGorry et al. [3] found that activation order of the extensor musculature at initiation of extension from a flexed posture progressed from caudal to cephalic in healthy subjects. Previous work aimed at identification of predisposing factors for LBP development during a standing exposure in previously asymptomatic people, showed that people who developed LBP during standing had significantly greater relaxation of the gluteus maximus muscles compared to their non-pain developing counterparts [4]. The purpose of the present study was to investigate differences in extensor muscle recruitment during the return to stand (RTS) phase of standing full flexion between this same sample of pain developers (PD) and non-pain developers (NPD).

METHODS

These analyses were conducted on data collected as part of a larger study at the University of Waterloo. Forty-three participants (22 male, 21 female) with no previous history of LBP had surface electromyography (EMG) collected from 3 bilateral trunk and hip muscles, including thoracic erector spinae (TES), lumbar erector spinae (LES) and gluteus maximus (GMax). Participants performed standing full flexion trials prior to a 2 hour standing exposure. Visual Analog Scale (VAS) for LBP was completed every 15 minutes during the 2 hours of standing. Participants were categorized as PD/NPD based on a cutoff threshold of > 10 mm on VAS. EMG data were windowed to isolate the RTS phase of the full flexion movement. Cross-correlation analyses were performed on the linear enveloped data to determine phase relationships (phase lag at peak correlation) between GMax, LES, and TES muscles. Data were entered into the cross-correlation equation such that a +ve phase lag indicated the first-listed muscle in a pair was activated first and a -ve phase lag indicated the first-listed muscle in a pair was activated second. Paired t-tests were performed on phase lag data for right/left muscle pairs, and when no differences were found (p > 0.05) these data were averaged to yield a single measure for the muscle group. Phase lag data were entered into a 2–way ANOVA with factors of PD/NPD group and gender with significance criterion set at p <0.05.

RESULTS AND DISCUSSION

Twenty-six and 17 participants were classified as NPD and PD respectively. There were no significant differences detected between left/right muscle pairs, therefore symmetry was assumed. NPD individuals demonstrated the previously reported pattern of caudal to cephalic muscle recruitment during RTS. PD individuals demonstrated the opposite pattern of activation, with cephalic muscles being activated prior to more caudal muscles (Figure 1).

There was a main effect of PD/NPD group for phase lag between LES and GMax ($F_{1,39} = 5.22, p = 0.03$), with PD activating GMax prior to LES (mean lag = $+ 0.038 \pm 0.33$ s) compared with NPD, where LES was activated prior to GMax (mean lag = $- 0.196 \pm 0.33$ s) (Figure 1). There were no
significant differences between NPD/PD groups detected for phase lag between the other muscle pairs (TES-LES, TES-GMax).

![Figure 1](image1.png)

**Figure 1.** NPD individuals activated more caudal muscles prior to more cephalic muscles, or a bottom up pattern, during RTS, while PD showed the reverse pattern. The PD group activated GMax significantly later than LES during RTS ($p < 0.05$).

There was also a main effect of gender for phase lag between TES and GMax ($F_{1,39} = 4.42$, $p = 0.04$) with females activating TES prior to GMax (mean lag = -0.123 ± 0.46 s), and males activating GMax prior to TES (mean lag = +0.085 ± 0.50 s) (Figure 2). There were no significant differences between genders detected for phase lag between the other muscle pairs (TES-LES, LES-GMax). There were no significant interactions between PD/NPD group and gender.

![Figure 2](image2.png)

**Figure 2.** There were significant gender differences in activation of extensor musculature during return to stand for the TES-GMax pair with males activating the TES prior to GMax and females demonstrating the opposite sequence.

**CONCLUSIONS**

Individuals predisposed to LBP during standing demonstrated a reverse pattern of extensor muscle activation to accomplish return to stand from lumbar forward flexion compared with individuals who did not develop LBP during standing. This was significant for the LES-GMax pair, with PD individuals having delayed activation of the GMax. It is of interest that these individuals had no history of clinical LBP. Alterations in muscle activity have been found previously in people who are predisposed to LBP development during the functional task of standing, leading the authors to hypothesize that these individuals may constitute a sub-clinical group who could be at future risk for LBP development. This provides further support for altered baseline muscle activation patterns as predictive factors for identification of individuals at-risk for LBP development.

**REFERENCES**


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