INTRODUCTION

Low back pain (LBP) is a disabling, pervasive, and costly medical condition that affects up to 80% of the US population [1]. Although previous investigations demonstrated a link between muscle activity and LBP, the relationship is dependent on variables such as the movement type, posture, and diagnostic group [2]. The cause of low back pain is often multifactorial, so the relationship between pain and the muscles that stabilize the spine is of interest to both clinicians and researchers alike. Conflicting evidence exists regarding paraspinal muscle activity in patients with LBP. Hypertonicity is a generally accepted clinical correlate of low back pain; however, some studies have demonstrated decreased electromyographic (EMG) amplitude in participants with LBP when compared to healthy controls [2]. It is not clear how activation amplitude contributes to spine stability, and recent attention has been given to timing and coordination of paraspinal muscle activation. For instance, patients with chronic LBP have delayed response of various trunk muscles during activities such as task performance, spinal loading, and anticipation of limb movement [3].

The multifidus (MF) and erector spinae (ES) muscles are extensor muscles that contribute to lumbar spine stability. However, the principle action of the MF is posterior sagittal rotation on a segmental level [4] while the lever arm of ES may best contribute to broad motions. Rapid shoulder flexion produces moments on the spine that are counteracted by activity in the lumbar musculature. Recent investigations of paraspinal timing and coordination in preparation for self-generated arm movements indicate a movement dependent difference in timing between the ES and the MF in a healthy population [5]. However, it is not clear whether this pattern of timing in the paraspinal musculature is similar in the presence of LBP. The objective of this investigation was to assess the differences in MF and ES activation timing during rapid arm flexion in participants with and without LBP. We hypothesized that onset of MF activity would occur before ES in both groups.

METHODS

Two males and nine females participated in this investigation, seven without a history of LBP and four with LBP at the time of testing verbally rated between two and seven (out of ten). EMG activity was recorded from the MF and ES at the L2 spinal level during voluntary shoulder flexion with fine wire electrodes [6] and surface electrodes, respectively. A single motion capture marker was placed on the wrist and used to calculate the onset timing of arm flexion.

With feet shoulder width apart, participants performed rapid shoulder flexion movements from neutral to approximately 60 degrees from vertical. Initiation of the movement immediately followed verbal cues of “Ready?” followed 1-2 seconds later by “Go!”. Participants were instructed that speed was the primary goal of the movement.

EMG signals were sampled at 2000 Hz and bandpass filtered (4th order Butterworth, 15-350Hz) then transformed using the Teager-Kaiser Energy operator [7]. Data were full-wave rectified and filtered (4th order low pass Butterworth, 50Hz cutoff) to create a linear envelope. EMG onset was defined as exceeding a threshold of baseline plus 15 standard deviations 200ms before or after onset of arm movement (peak acceleration of wrist marker). All onset timings were verified by visual inspection.
Differences in muscle activation onsets between muscles (MF, ES) were tested using one-directional paired t-tests within each group (Healthy, LBP). Level of significance was set at $\alpha=0.05$. Effect sizes for each comparison were assessed using Cohen’s $d$.

RESULTS AND DISCUSSION

Muscle activation onsets in both the MF and ES occurred before onset of the arm flexion (Figure 1) and the mean activation onset ranged between 64.8msec and 105.3msec before arm flexion (Table 1). In the healthy group, the MF onset occurred 30.2±21.6% earlier than ES onset ($p=0.025$, $d=1.19$). In the LBP group, there was no difference between MF and ES onset ($p=0.123$, $d=0.334$).

Table 1: Mean ± SD of EMG onset (msec) preceding peak arm acceleration.

<table>
<thead>
<tr>
<th>Group</th>
<th>MF</th>
<th>ES</th>
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<tr>
<td>Pain</td>
<td>74.5 ± 33.0</td>
<td>64.8 ± 24.7</td>
</tr>
<tr>
<td>Healthy</td>
<td>105.3 ± 43.4</td>
<td>68.0 ± 12.7</td>
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Earlier onset of MF activity during rapid arm flexion occurred in the healthy group, but not in the pain group. While standing, rapid arm flexion creates a flexion moment on the spine that is stabilized by an extension moment produced by the paraspinal musculature. Previous findings in healthy participants demonstrated earlier activation of the deep fibers of the MF compared to the ES in an extension activity [5]. This investigation confirms this coordination in the healthy group with a flexion movement, but not the LBP group. Earlier onset of the MF indicates an anticipatory role of the MF and provides evidence for stabilizing function role of the MF that may take advantage of compressive action when activated.

Mean ES activity for both the pain and the healthy group were similar (64.8msec, 68.0msec), which indicates that earlier onset of MF activity in the healthy group was responsible for differences found in this investigation. It unclear what role MF/ES coordination plays in spinal stability or how MF dysfunction in patients with chronic LBP may affect this relationship. Underlying causes of MF dysfunction may be related to several factors such as pain inhibition or fatty infiltration that accompanies LBP. [8] The timing difference found in this study is juxtaposed with the measures of EMG amplitude and clinical hypertonicity and suggests the importance of further examination of MF activation timing in various populations and tasks.

In summary, anticipatory onset of MF and ES activity measured during rapid arm flexion in healthy participants are different in healthy participants but not in LBP participants. It remains unclear what role activation timing plays in spine stability. Additional investigations of paraspinal muscle timing and coordination may provide insight into healthy and pathologic mechanisms of motor control in spine stability.

REFERENCES


AKNOWLEDGEMENT

This work was supported in part by grant R00 AT004983-03 (to BSD) from NIH (NCCAM).