Altered Trunk Muscle Recruitment in People With Low Back Pain With Upper Limb Movement at Different Speeds

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Objective: To compare trunk muscle coordination in people with and without low back pain with varying speeds of limb movement.

Study Design: Abdominal and back extensor muscle activity in association with upper limb movement was compared among three speeds of movement and between people with and without low back pain.

Participants: Fourteen subjects with a history of recurrent low back pain and a group of age- and sex-matched control subjects.

Measures: The onsets of electromyographic activity of the trunk and limb muscles, frequency of trunk muscle responses, and angular velocity of arm movements.

Results: Early activation of transversus abdominis (TrA) and obliquus internus abdominis (OI) occurred in the majority of trials, with movement at both the fast and intermediate speeds for the control group. In contrast, subjects with low back pain failed to recruit TrA or OI in advance of movement with fast movement, and no activity of the abdominal muscles was recorded in the majority of intermediate speed trials. There was no difference between groups for slow movement.

Conclusion: The results indicate that the mechanism of preparatory spinal control is altered in people with lower back pain for movement at a variety of speeds.

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INTEREST IN EVALUATING changes in recruitment of trunk muscles in people with low back pain (LBP) and the effect that these changes may have on the function of the spine is increasing.1-6 Recent experimental evidence suggests that people with a history of LBP have altered recruitment of specific trunk muscles in response to spinal disturbances.4-7,12 This change in recruitment has only been observed in people with a history of LBP in a more functionally relevant task.3,5 Because of the inherent instability of the spine, particularly in the neutral position,8 these changes in recruitment may indicate inadequate protection of the spinal structures from injury.

When perturbing forces act on the spine, precise temporal and spatial recruitment of the trunk muscles is essential to adequately protect the spine.9 By investigation of the response of the trunk muscles to a perturbation to the spine resulting from voluntary limb movement, it is possible to investigate the strategy used by the central nervous system (CNS) to prepare the spine. When a limb is moved, the configuration of the body is altered and reactive forces are imposed on the body that are equal in magnitude but opposite in direction to those producing the movement.10 Thus, when a shoulder is flexed, reactive forces act backwards and downwards on the spine.11,12 This activity must be preprogrammed by the CNS because it occurs in advance of the onset of activity of the muscle responsible for limb movement (ie, "feedforward").13 When people have LBP, however, the contraction of TrA and OI is delayed and therefore absent from the period preceding the onset of movement.1

This change in trunk muscle recruitment has only been identified with movement performed rapidly. Movements are rarely performed at such speeds during normal function, however. The aim of the present study was to investigate the activation of the trunk muscles with movements at slower speeds to evaluate whether changes in recruitment would be observed in people with a history of LBP in a more functionally relevant task.

METHODS

Subjects

Twenty-eight subjects participated in the study, including 14 (seven men, seven women) with a history of LBP and a group of age- (±3yrs) and sex-matched control subjects. The mean ± SEM age, height, and weight of the LBP subjects were 30 ± 2 years, 1.74 ± .02 m, and 63 ± 8 kg, respectively. Strict criteria were used for selection of the subjects for the LBP group. Subjects must have had insidious-onset LBP of at least 18 months' duration for which they had sought medical or allied health intervention and that had caused them to be absent from work a minimum of 3 days. The subjects must have had at least one episode of back pain per year or have semicontinuous back pain. The mean duration of symptoms was 8.3 years (range, 2 to 30 yrs). The subjects were to be pain free at the time of testing and were medically screened to rule out back pain of nonmusculoskeletal origin.

Control subjects had no history of LBP that had limited function or for which they had sought medical or allied health intervention. The mean age, height, and weight of the control subjects were 29 ± 2 years, 1.72 ± .03 m and 66 ± 3 kg, respectively. There was no significant difference between subject groups for any of the demographic parameters (p < .05).

Subjects were excluded from either group if they had neurologic symptoms, observable spinal deformity (eg, scoliosis), previous lumbar surgery, neuromuscular or joint disease, or...
if they had undertaken abdominal or back muscle training in the 3 months before testing. Habitual physical activity was measured using a questionnaire developed and validated by Baekke and colleagues. The mean habitual activity scores for work, sport, and leisure were 8.92 ± 0.39 and 8.45 ± 0.25 for the LBP and control groups, respectively. These scores indicate a high level of physical activity for both groups and there was no statistical significance between the groups (p < .05). The study was approved by the Institutional Medical Research Ethics Committee.

**Electromyographic Recordings**

Fine-wire and surface electromyography (EMG) electrodes were used. Ag/AgCl surface electrodes were placed over the anterior portion of the right deltoid, over the left rectus abdominis (RA) 2 cm lateral to the midline below the umbilicus and over the left ES 1 cm lateral to the LA-5 interspace. Although the electrode for ES was primarily placed over the superficial fibres of multifidus, it was expected that this placement would be subject to considerable cross talk from the adjacent muscles and would be best referred to as general ES EMG rather than specifically from multifidus. Surface electrodes were placed in parallel with the muscle fibres with an interelectrode distance of 32mm.

Bipolar fine-wire electrodes were fabricated from Teflon-coated stainless-steel wire (75μm diameter) inserted into a hypodermic needle (.70 × 32mm). Approximately 1mm of Teflon was removed from the tips and the wires were bent back by 1 and 3mm. Electrode insertion into the left abdominal muscles was performed under the guidance of ultrasound imaging using a 5MHz transducer. Electrodes were inserted into TrA 2cm medial to the proximal end of a line drawn vertically from the anterior superior iliac spine to the caudal border of the rib cage, OI 2cm medial and superior to the anterior superior iliac spine, and obliquus abdominis externus (OE) midway between the iliac crest and distal border of the rib cage in the mid axillary line. Because of the close proximity of the three abdominal muscles, it is possible that the signals could be contaminated by cross-talk from adjacent muscles; therefore, several procedures were undertaken to confirm the precision of the recordings. Accuracy of electrode placement was confirmed by observation of movement of the wires with ultrasound imaging during gentle traction of the wires. In addition, subjects performed a series of manoeuvres aimed at specifically activating the muscles. This involved ipsilateral trunk rotation for OI and contralateral rotation for OE. Because it is difficult to perform a maneuver that isolates the activity of TrA, the accurate location of this electrode was confirmed if the onset of EMG recorded with this electrode was consistently different from that for the other adjacent muscles, thus excluding the possibility of cross talk. Finally, the possibility for cross-talk contamination from adjacent muscles was reduced by the small detecting area of the electrodes.

**Limb Movement Speed Evaluation**

The speed of limb movement was evaluated using a Position-Velocity Transducer to measure the linear velocity of distraction of a cable attached to the arm 0.5m from the tip of the acromion. The linear velocity was converted to angular velocity using trigonometry. Because of the slight resistance provided by the distraction of the cable at the initiation of movement, the kinematic evaluation was conducted separately from the EMG trial, and on five subjects selected randomly from each group. This was necessary because feedforward postural responses are modified when limb movement is performed against resistance. The mean and peak angular velocities of limb movement were calculated for analysis.

**Procedure**

Subjects performed right shoulder flexion at three different speeds while standing on a force platform that provided auditory feedback of inequality of mediolateral weight bearing. This feedback was used to ensure that weight shifting did not occur before performance of the movement. Movement was performed in response to a visual stimulus that was preceded by a warning stimulus by a random period of between 0.5 to 4 seconds. No pacing guide was used to control the movement speed because behavioural constraint produces variation in the temporal sequence of postural muscle contraction. Thus, three broad categories of movement speed were used that correspond to definitions reported previously. The speeds were as follows: (1) *Fast*: Movement performed as rapidly as possible. Subjects were verbally encouraged throughout the procedure to achieve their maximum possible arm movement speed with emphasis on speed of movement and not the accuracy of the distance moved. (2) *Intermediate*: Movement performed at a speed natural to the subject. This condition involved movement at a self-paced comfortable speed to point towards the stimulus to move. (3) *Slow*: Movement at a slow speed completing 60° of movement in approximately 2 seconds. This speed of movement was demonstrated to the subject by the examiner.

Subjects were allowed five practice repetitions at each speed. Ten consecutive repetitions of each movement were performed and the order of movements was randomized. Pilot studies showed that the trunk muscle responses were consistent over a wide range of speeds, satisfying the criteria for each condition. This indicated that no further control of the limb velocity was necessary. The evaluation of limb movement speed (n = 5) was undertaken in an identical manner on a different day from the EMG trial. Efforts were made to ensure that the movement was identical between days by using identical instructions and guidance by the examiner. Subjects performed 10 repetitions of shoulder flexion at each of the three movement speeds.

**Data Analysis**

EMG data were sampled at 2,000Hz, bandpass filtered between 20 and 1,000Hz, 12-bit analog-to-digital converted, and stored on computer for analysis. Following full-wave rectification and low-pass filtration at 60Hz, the onsets of EMG were identified using Matlab® software as the point where the mean of the 50 subsequent samples exceeded the background level of activity by two standard deviations. The EMG onsets identified in this manner have been compared to visually determined values and found to be accurate. All EMG onsets were checked visually to ensure that they were valid and not interrupted by artifact from movement or the ECG.

The reaction time (from stimulus to move to onset of EMG) and the latency between the onset of EMG of deltoid and each of the trunk muscles formed the basis of the analysis. The trunk muscle activity was regarded as "feedforward" if its onset occurred between 100msec before to 50msec after that of deltoid. This period provides insufficient time for a response of the trunk muscles to be mediated as a result of even the fastest monosynaptic reflex. Correspondingly, any trunk muscle response occurring in this period must be initiated as part of the motor command for limb movement or in parallel with the motor command for limb movement. The reaction time, latency between the onset of deltoid EMG and that of each of the trunk muscles, and the mean and peak angular velocity were compared between movement speed conditions using a repeated-
measures analysis of variance (ANOVA) and Duncan’s multiple range test. The frequency of response of the trunk muscles was compared among speeds of movement and between groups using Student’s t test. Significance was set at \( \alpha = .05 \).

All analyses were performed on EMG onsets identified from single trials and then averaged. However, the results of the analysis were not altered when the raw rectified traces were averaged before detection of EMG onset (figs 1 and 2).

RESULTS

Variation in Limb Movement Speed

The mean and peak angular velocity of limb movement was not different between subject groups for movement at the fast and intermediate speeds (table 1). Thus, any difference between the subject groups cannot be explained by variation in this parameter. In contrast, the LBP subject group had a faster mean and peak velocity than the control group at the slow limb movement speed.

Control Group Neuromotor Response

When movement of the upper limb was performed in response to a visual stimulus to move, the reaction times of deltoid and the trunk muscles, except RA, were increased in the slow condition in comparison with either the fast or intermediate movement speeds (fig 1) (deltoid: \( F(4,3) = 9.31, p < .05 \); TrA: \( F(14,3) = 14.02, p < .05 \); OI: \( F(14,3) = 13.07, p < .05 \); OE: \( F(14,3) = 23.69, p < .001 \); ES: \( F(14,3) = 24.27, p < .001 \)). There was no difference in reaction time between the fast and intermediate condition for any muscle. The latency between the onset of deltoid EMG and that of the trunk muscles in the slow condition was different from the fast and intermediate speed movements. Again, there was no difference between the intermediate and fast conditions (tables 2 and 3; figs 1 and 3).
The onsets of TrA, ES, and OI EMG occurred within the "feedforward" criteria for the fast condition. In contrast, only TrA and ES occurred with in the "feedforward" criteria in the intermediate condition. No onset of trunk muscle EMG was recorded within the "feedforward" criteria for the slow speed condition (except RA, which was active in only one subject; fig 3).

EMG response of the trunk muscles was identified in only a small number of trials of movement performed at the slow speed with a frequency of response that was significantly less than for the other two conditions (fig 4). The frequency of response ranged from 0.7% for RA to 58% for ES. However, there was no significant difference between the fast and intermediate conditions, with the exception of RA and OE, which were inactive in 45% and 75% of the trials at the intermediate speed, respectively.

**LBP Neuromotor Response**

Similar to the control group, the reaction times of deltoid, TrA, OE, and ES increased with decreasing limb movement speed (deltoid: F(2,14) = 13.81, p < .001; TrA: F(2,14) = 10.48, p < .001; OE: F(2,14) = 4.98, p < .05; ES: F(2,14) = 36.26, p < .001). Unlike the control group, the intermediate and fast movement conditions were different for all muscles except deltoid (fig 2). The changes in latency between the onset of deltoid EMG and that of the trunk muscles between movement speeds was identical to the changes in reaction time, except there was no difference between the intermediate and fast conditions for ES (table 2; fig 2 and 3). In contrast to the control group, only the onset of ES occurred within the "feedforward" criteria in the fast and intermediate speed conditions. Notably, the onset of TrA was delayed in both the fast and intermediate movement conditions. Although the onset of RA also occurred within the "feedforward" criteria for both the fast and intermediate conditions, the response was only present in less than 4% of trials for the intermediate speed condition.

Although the subjects with a history of LBP performed the slow movement at a faster average angular velocity than the controls, the response of the trunk muscles (except ES) occurred with a frequency that was not significantly different between the groups (p > .05; fig 4). In contrast to the control group, responses of the trunk muscles occurred significantly less frequently with movement performed at an intermediate speed that at the fast speed for all muscles (p < .05). In addition, the frequencies of responses of all of the trunk muscles (except ES) at this speed were significantly less for the LBP group compared to the control subjects. Responses of all trunk muscles were recorded frequently for movement at the fast speed and were not significantly different from the control subjects.

**Intergroup Comparison**

The onsets of EMG of TrA (F(1,14) = 31.47, p < .001) and OI (F(1,14) = 4.45, p < .05) were delayed in the LBP group in comparison with the control group for movement at the fast speed (fig 3). In addition, the onset of TrA was delayed in the LBP group with movement at the intermediate speed (F(1,14) = 18.53, p < .001). There was no other difference in EMG onsets of movement with this speed. There was no difference in latency or reaction time between the two subject groups for movement at the slow speed.

**DISCUSSION**

The results of the present experiment indicate that trunk muscle recruitment associated with limb movement is altered in people with a history of LBP with movement at a variety of speeds. This change in recruitment presented as delayed onset of contraction of TrA and OI with movement at the fast speed and a profound absence of contraction of all of the abdominal muscles with movement at the intermediate speed. In the small number of trials in which a response of TrA was recorded for the LBP group with movement at the intermediate speed, it was delayed compared with that of the control subjects. These changes are unlikely to be attributed to variation in limb movement speed because no difference in limb movement speed was identified between subject groups. These discrepancies in the postural response of the trunk muscles provide evidence for a

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**Table 1: Angular Velocity of Shoulder Flexion for Control and LBP Subjects (mean ± SEM) Measured in a Trial Separate from the EMG Data Using an Identical Procedure (n = 8)**

<table>
<thead>
<tr>
<th>Speed/Parameter</th>
<th>Non-LBP</th>
<th>LBP</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fast Peak velocity</td>
<td>514.9 ± 56.5</td>
<td>498.4 ± 41.2</td>
<td>.06</td>
</tr>
<tr>
<td>Mean velocity</td>
<td>308.4 ± 32.6</td>
<td>285.7 ± 23.8</td>
<td>.32</td>
</tr>
<tr>
<td>Intermediate Peak velocity</td>
<td>258.8 ± 16.1</td>
<td>294.9 ± 17.0</td>
<td>.98</td>
</tr>
<tr>
<td>Mean velocity</td>
<td>144.2 ± 15.4</td>
<td>159.9 ± 11.6</td>
<td>.86</td>
</tr>
<tr>
<td>Slow Peak velocity</td>
<td>67.2 ± 10.4</td>
<td>92.5 ± 8.9</td>
<td>3.45&lt;sup&gt;*&lt;/sup&gt;</td>
</tr>
<tr>
<td>Mean velocity</td>
<td>35.8 ± 4.3</td>
<td>53.8 ± 3.2</td>
<td>6.76&lt;sup&gt;*&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>*</sup> p < .05.

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**Table 2: Reaction Time of Deltoid EMG and Each Trunk Muscle (mean ± SEM) Showing the Results of the ANOVA Comparing Latency for Muscles Between Speeds**

<table>
<thead>
<tr>
<th>Non-LBP</th>
<th>Deltoid</th>
<th>TrA</th>
<th>OI</th>
<th>OE</th>
<th>RA</th>
<th>ES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fast</td>
<td>188 ± 9</td>
<td>149 ± 9</td>
<td>216 ± 18</td>
<td>246 ± 15</td>
<td>271 ± 17</td>
<td>196 ± 11</td>
</tr>
<tr>
<td>Natural</td>
<td>177 ± 17</td>
<td>202 ± 26</td>
<td>260 ± 34</td>
<td>325 ± 34</td>
<td>460 ± 83</td>
<td>194 ± 18</td>
</tr>
<tr>
<td>Slow</td>
<td>309 ± 42</td>
<td>646 ± 129</td>
<td>753 ± 144</td>
<td>887 ± 102</td>
<td>142&lt;sup&gt;*&lt;/sup&gt;</td>
<td>422 ± 44</td>
</tr>
<tr>
<td>F</td>
<td>9.31&lt;sup&gt;*&lt;/sup&gt;</td>
<td>14.02&lt;sup&gt;*&lt;/sup&gt;</td>
<td>13.07&lt;sup&gt;*&lt;/sup&gt;</td>
<td>23.69&lt;sup&gt;*&lt;/sup&gt;</td>
<td>4.45&lt;sup&gt;*&lt;/sup&gt;</td>
<td>24.27&lt;sup&gt;*&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LBP</th>
<th>Deltoid</th>
<th>TrA</th>
<th>OI</th>
<th>OE</th>
<th>RA</th>
<th>ES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fast</td>
<td>188 ± 14</td>
<td>313 ± 25</td>
<td>270 ± 22</td>
<td>291 ± 14</td>
<td>307 ± 24</td>
<td>200 ± 13</td>
</tr>
<tr>
<td>Natural</td>
<td>257 ± 22</td>
<td>476 ± 45</td>
<td>427 ± 64</td>
<td>414 ± 31</td>
<td>223 ± 33</td>
<td>298 ± 34</td>
</tr>
<tr>
<td>Slow</td>
<td>417 ± 61</td>
<td>500&lt;sup&gt;1&lt;/sup&gt;</td>
<td>585 ± 111</td>
<td>837 ± 65</td>
<td>472&lt;sup&gt;2&lt;/sup&gt;</td>
<td>580 ± 63</td>
</tr>
<tr>
<td>F</td>
<td>13.81&lt;sup&gt;1&lt;/sup&gt;</td>
<td>10.48&lt;sup&gt;1&lt;/sup&gt;</td>
<td>2.86</td>
<td>4.98&lt;sup&gt;1&lt;/sup&gt;</td>
<td>3.29</td>
<td>36.16&lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>1</sup> p < .05.
<sup>2</sup> p < .001.
<sup>1</sup> Denotes that contraction occurred in only one subject and therefore no standard deviation is shown.
change in the strategy used by people with a history of LBP for protection and control of the spine.

**Normal Recruitment of the Trunk Muscles Associated with Limb Movement**

The sequence of trunk muscle recruitment identified in the present experiment with movement at the fast speed is consistent with previous studies indicating early “feedforward” activation of ES, 16,28,29 TrA, and OI. 1,14 This trunk muscle activity is considered to contribute to either control of the reactive forces resulting from limb movement, control of the displacement of the center of mass resulting from changes in the configuration of the body,12,15,30 or a combination of these disturbances. Correspondingly, it has been reported recently that a small magnitude trunk extension (<2°) results from the “feedforward” activation of ES, which is in the direction opposite to both the reactive forces resulting from shoulder flexion and displacement of the centre of mass generated by forward arm movement.12,15 In contrast to ES, the horizontally arranged muscle fibres of TrA and the lower portion of OI do not have a mechanical advantage to directly control reactive forces at the spine.31 In addition, there is debate regarding the ability of TrA to produce trunk rotation.19,20 Alternatively it has been suggested that TrA may contribute to stability of the spine and pelvis in a non-direction-specific manner by either increasing the pressure in the abdominal cavity20,32 or by increasing the tension in the thoracolumbar fascia.33

The variation in trunk muscle response between the slow and two faster movement speeds is in agreement with previous investigations, which identified a consistent postural response for movements performed above a threshold velocity, whereas responses below that velocity are variable and often ab-

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**Table 3: Latency Between Onset of Deltoid EMG and for Each Trunk Muscle (mean ± SEM) Showing Results of the ANOVA Comparing Latency for Muscles Between Speeds**

<table>
<thead>
<tr>
<th></th>
<th>TrA</th>
<th>OI</th>
<th>OE</th>
<th>RA</th>
<th>ES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Non-LBP</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fast</td>
<td>$-39 \pm 8$</td>
<td>$26 \pm 14$</td>
<td>$57 \pm 13$</td>
<td>$85 \pm 16$</td>
<td>$9 \pm 9$</td>
</tr>
<tr>
<td>Natural</td>
<td>$32 \pm 21$</td>
<td>$76 \pm 18$</td>
<td>$143 \pm 40$</td>
<td>$291 \pm 78$</td>
<td>$22 \pm 9$</td>
</tr>
<tr>
<td>Slow</td>
<td>$406 \pm 146$</td>
<td>$359 \pm 96$</td>
<td>$347 \pm 113$</td>
<td>$34^*$</td>
<td>$151 \pm 36$</td>
</tr>
<tr>
<td>$F$</td>
<td>8.60*</td>
<td>9.29*</td>
<td>7.26*</td>
<td>5.32*</td>
<td>12.43*</td>
</tr>
<tr>
<td><strong>LBP</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fast</td>
<td>$126 \pm 28$</td>
<td>$80 \pm 22$</td>
<td>$92 \pm 11$</td>
<td>$124 \pm 25$</td>
<td>$11 \pm 8$</td>
</tr>
<tr>
<td>Natural</td>
<td>$260 \pm 43$</td>
<td>$147 \pm 43$</td>
<td>$134 \pm 30$</td>
<td>$15 \pm 26$</td>
<td>$34 \pm 14$</td>
</tr>
<tr>
<td>Slow</td>
<td>$306^*$</td>
<td>$145 \pm 71$</td>
<td>$521 \pm 40$</td>
<td>$-28^*$</td>
<td>$152 \pm 36$</td>
</tr>
<tr>
<td>$F$</td>
<td>22.23*</td>
<td>0.48</td>
<td>0.39*</td>
<td>2.25</td>
<td>9.82*</td>
</tr>
</tbody>
</table>

* $p < .05$.  
† $p < .001$.  
* Denotes that contraction occurred in only one subject and therefore no standard deviation is shown.
Increased stress on the passive spinal structures has been found this threshold velocity to be approximately 70°/sec to 100°/sec for ES, which falls between the intermediate and slow speeds used in the present experiment. The velocity dependence of trunk muscle recruitment has implications for the function performed by the postural muscle activity. Variation in limb movement speed alters the magnitude of reactive forces because of variation in the limb acceleration. Thus, components of the trunk muscle response associated with the control of reactive forces should vary between movement speeds. In contrast, displacement of the center of mass as a result of altered body configuration is unchanged by variation in movement speed. The failure of TrA and the other abdominal muscles to respond in the majority of trials with movement at the slow speed suggests a contribution to the control of the reactive forces. It has been suggested that the passive viscoelastic properties of the tissues are sufficient to maintain stability when the perturbing force is small.

In contrast to the abdominal muscles, the frequency of response of ES was present in the majority of trials at the slow speed of limb movement. The presence of response of ES at the slow speed is indicative of a contribution to control of the displacement of the center of mass resulting from altered limb geometry. The delayed onset of ES activity with slow movement compared to the fast and intermediate speed conditions is most likely due to the increased latency for the limb to reach a position where contraction of the trunk extensor is required. Thus, at slower speeds of limb movement, recruitment of ES is sufficient to control postural stability. As the speed of limb movement is increased, however, recruitment of ES occurs in a "feedforward" manner to contribute to the anticipatory control of the reactive forces (by initiating trunk motion in the opposite direction) in addition to the changes resulting from altered body configuration.

Abnormal Recruitment of the Trunk Muscles Associated With Limb Movement in People With LBP

In comparison with the control subjects, the EMG onsets of TrA and OI were delayed in the subjects with a history of LBP with movement at the fast speed. This finding is consistent with previous results. Because of this change in the preprogrammed pattern of muscle recruitment, ES was the only recorded trunk muscle active within the "feedforward" criteria. This finding indicates that preparatory processes still occur in the central nervous system to initiate a sequence of trunk muscle activity before limb movement. Although trunk movement was not recorded in the present investigation, the presence of the "feedforward" contraction of ES suggests that preparatory trunk motion may occur in a similar manner as previously reported for control subjects. However, the component of spinal stability controlled by TrA and OI may be different. When people with a history of LBP moved their upper limb at the intermediate speed, contractions of all of the abdominal muscles were absent in the majority of trials. On the basis of the findings of the control group and the previous studies, this would suggest an increase in the threshold velocity required to produce a trunk muscle response.

The pattern of trunk muscle recruitment with movement at the slow speed was identical for both subject groups. As mentioned previously, the major perturbing factor with limb movement at the slow speed is the displacement of the center of mass due to the forward movement of the arm. Correspondingly, no change in ES onset or frequency of recruitment was identified between the control and LBP groups at any limb movement speed. Thus, the results suggest that control of the center of mass was unaffected in LBP patients. However, the accuracy of control of the center of mass may not be as exact in people with LBP.

The present results indicate that the control of the reactive forces is the factor principally affected in the presence of LBP. The consequence of this change in control was not measured. However, in the neutral position minimal restraint is provided by passive structures and stability of the spinal segments is dependent on the contraction of the surrounding muscles. The identified changes in the muscular control of the trunk in people with a history of LBP may potentially expose the spinal structures to an increased risk of microtrauma and injury.
identified, using a computer simulation, to result from decreased contribution of the muscle system to spinal stability. Indirect support for the relationship between TrA activity and increased contribution of the muscle system to spinal stability has come from the results of a recent randomized, controlled clinical trial that indicated a reduction and pain and disability in a group of chronic LBP patients as a result of TrA training. This finding needs further clarification however, as this study did not confirm that the positive outcome was caused by a change in TrA function.

While the majority of research investigating the function of the trunk muscles associated with LBP has focused on the strength and endurance of the trunk muscles, few studies have investigated the recruitment patterns of trunk muscles in people with LBP. Other changes in trunk muscle control include a decrease in cocontraction of the ES with the abdominal muscles during performance of a sit-up maneuver, a reduced ratio of OI activity in comparison with that of RA during the performance of a voluntary abdominal muscle contraction, absence of ES relaxation at the end of range of trunk flexion in standing, and a loss of the biphasic pattern of ES recruitment when catching an unexpected load anteriorly in the hands. Although none of the previous studies are directly comparable with the present findings, they do indicate that changes in motor control are a consistent finding in association with LBP. The mechanism of these changes in motor control has not been addressed in the literature. However, the only previous populations of subjects in which changes in “feedforward” muscle activation have been identified have been people with frontal lobe trauma and Parkinson disease. Whether the identified changes represent the outcome of changes in motor control and planning in the central nervous system requires further evaluation.

The results of the present investigation have implications for clinical management of patients with LBP. First, the identification of changes in coordination of the trunk muscles (particularly TrA and OI) supports previous findings and provides evidence that therapeutic exercise should address changes in control rather than strength training. Approaches aimed at retraining trunk control have been described and have been evaluated in a randomized, controlled clinical trial with good results. Second, the results provide evidence that coordination of the trunk muscles is altered in people with a history of LBP even when they are without pain.

CONCLUSIONS

Movement of the upper limb by people with a history of LBP is associated with changes in the contraction of the abdominal muscles at both intermediate and fast movement speeds. It is important to consider that a person with a history of LBP without current symptoms may be at greater risk of reinjury because of inadequate muscular stabilization of the spine.

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References


Suppliers

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