Inefficient Muscular Stabilization of the Lumbar Spine Associated With Low Back Pain
A Motor Control Evaluation of Transversus Abdominis

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Study Design. The contribution of transversus abdominis to spinal stabilization was evaluated indirectly in people with and without low back pain using an experimental model identifying the coordination of trunk muscles in response to a disturbance to the spine produced by arm movement.

Objectives. To evaluate the temporal sequence of trunk muscle activity associated with arm movement, and to determine if dysfunction of this parameter was present in patients with low back pain.

Summary of Background Data. Few studies have evaluated the motor control of trunk muscles or the potential for dysfunction of this system in patients with low back pain. Evaluation of the response of trunk muscles to limb movement provides a suitable model to evaluate this system. Recent evidence indicates that this evaluation should include transversus abdominis.

Methods. While standing, 15 patients with low back pain and 15 matched control subjects performed rapid shoulder flexion, abduction, and extension in response to a visual stimulus. Electromyographic activity of the abdominal muscles, lumbar multifidus, and the contralateral deltoid was evaluated using fine-wire and surface electrodes.

Results. Movement in each direction resulted in contraction of trunk muscles before or shortly after the deltoid in control subjects. The transversus abdominis was invariably the first muscle active and was not influenced by movement direction, supporting the hypothesized role of this muscle in spinal stiffness generation. Contraction of transversus abdominis was significantly delayed in patients with low back pain with all movements. Isolated differences were noted in the other muscles.

Conclusions. The delayed onset of contraction of transversus abdominis indicates a deficit of motor control and is hypothesized to result in inefficient muscular stabilization of the spine. (Key words: abdominal muscles, low back pain, lumbar spine, movement, posture)

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Dysfunction of the ventral and dorsal muscles of the trunk have been studied in relation to low back pain (LBP) for many years. This has been based on the premise that insufficiency of muscle function leads to stress and undue load on the joints and ligaments of the spine. However, despite considerable effort, few studies of muscular strength and endurance have identified a discernible and consistent abnormality of trunk muscle function in people with LBP. Nevertheless, rehabilitation techniques advocating the training of muscular stabilization of the spine are purported to be effective, indicating that additional components of the muscular system of the lumbar spine require investigation. Although, many muscles of the trunk are capable of contributing to the stabilization and protection of the lumbar spine, recent evidence has suggested that transversus abdominis (TrA) may be critically involved and has been the focus of rehabilitation. Evaluation of the function of this muscle in people with LBP has not been addressed.

The muscle fibers of TrA run horizontally around the abdomen, attaching via the thoracolumbar fascia to the transverse processes of each of the lumbar vertebrae. The increase in intra-abdominal pressure and tensioning of the thoracolumbar fascia resulting from contraction of this muscle initially were thought to decrease spinal loading through the production of a trunk extensor moment. This theory has largely been refuted and consequently, it has been hypothesized that the contraction of TrA may enhance stabilization of the spine.

Farfan and McGill and Norman suggested that the contraction of the hoop-like TrA creates a rigid cylinder, resulting in enhanced stiffness of the lumbar spine. Similarly, it may be expected that lateral tension through the transverse processes of the lumbar vertebrae would
result in limitation of translational and rotational motion of the spine. Furthermore, the creation of a pressurized visceral cavity anterior to the spine results in the production of a force against the apex of the lumbar lordosis, which has been suggested to increase the stability of the structure of the spine for a variety of postures and movements.\textsuperscript{2} The unexpected continuous activation of TrA throughout flexion and extension of the trunk\textsuperscript{16} is consistent with this proposal. The consequence of dysfunction of TrA would be suboptimal control of the spine against forces acting to challenge the integrity of the spine.

**Motor Control of Lumbar Spine Stability**

The aspect of trunk muscle function that has received only limited attention in LBP research is evaluation of the motor control mechanisms.\textsuperscript{4,5} A model for the evaluation of motor control strategies for stabilization of the spine would necessarily involve identification of the coordination and timing of contraction of muscles contributing to spinal stiffness generation. On the basis of the previous argument, TrA should be included.

Evaluation of the motor control of spinal stability is difficult because of the complex nature of the system enabling it to deal with constantly changing demands as a function of continual variation in internal and external forces.\textsuperscript{45} Evaluation of this system would be facilitated by identification of the muscular response to a controlled challenge to stability. Several studies have attempted to achieve this by investigation of the response of the spinal muscles to external loading of the trunk by having subjects either catch a weight in their hands\textsuperscript{28,31,37} or by adding weight to a harness over their shoulders,\textsuperscript{11} indicating a rapid and often excessive response of the trunk muscles to control the disturbing force. In trials in which the loading was expected by the subject, the response preceded the loading.\textsuperscript{11,31,32} Using this model, Cresswell et al\textsuperscript{12} reported TrA to precede not only the acceptance of the load but also the onset of the other trunk muscles, providing further support for the role of this muscle in spinal stabilization.

The muscular response to this type of external loading relies on the control of spinal stability against forces in which the magnitude and direction are uncertain until the load is accepted, necessitating a gross general response of the trunk muscles and revealing little of the specificity expected of the spinal stabilizing system. Evaluation of the control strategy using a model in which the exact nature of the disturbance or perturbation can be anticipated by the central nervous system would facilitate a more specific investigation of this complex system.

Evaluation of the response of the body to movement of a limb provides such a model. In association with the change in the position of the center of mass produced by the limb displacement, dynamic forces are transmitted to the body by the inertial reactions between the segments.\textsuperscript{16} Using this model, the influence of the reactive forces at the spine has been evaluated with unilateral and bilateral movement of the shoulder\textsuperscript{61} and elbow.\textsuperscript{16} These studies either have calculated or recorded the production of a net flexion moment accompanying upper limb flexion and the converse with extension. Furthermore, unilateral limb movement also would be associated with a rotary moment. Accordingly, this disturbance to the stability of the spine and position of the center of mass of the body is consistent and may be predicted by the central nervous system.\textsuperscript{7,16,25}

Many studies have used this model for evaluation of control of body equilibrium and have indicated that the body deals with these predictable disturbances by activation of lower limb muscles before the initiation of the movement.\textsuperscript{3} Contraction of muscles in this way has been termed *anticipatory*.\textsuperscript{1} In the course of evaluating equilibrium control, several studies have identified contraction of rectus abdominis (RA) and erector spinae (ES) before the initiation of upper limb movement\textsuperscript{1,16,61}, suggesting that spinal control may be dealt with in an anticipatory manner. It is important that the contribution of muscles such as TrA be included in the evaluation of the muscular contribution to control of the lumbar spine against self-generated perturbation and that the results compared with those of a population with LBP.

**Purpose of the Study**

The objective of this research was to evaluate several hypotheses regarding the motor control of TrA and stabilization of the lumbar spine. Namely, it was hypothesized that contraction of TrA would precede the initiation of limb movement and the other muscles of the trunk (consistent with the work of Cresswell et al).\textsuperscript{11} Furthermore, considering the proposed mechanisms through which TrA may contribute to stabilization of the spine, it was predicted that the response of this muscle would not be influenced by changes in the direction of the reactive forces. Finally, it was hypothesized that people with LBP would have disruption of this spinal stabilizing mechanism, which would fail to contract TrA before movement. Preliminary results have been published as an abstract,\textsuperscript{20} and results of a preliminary study of a separate group of subjects without LBP is in preparation.

**Methods**

**Subjects.** Thirty subjects participated in the study including 15 patients (eight male, seven female) with a history of lumbar spine pain and 15 age- and sex-matched control subjects. All patients with LBP were medically screened to rule out back pain of nonmusculoskeletal etiology. Recruitment of the subjects with LBP was based on strict clinical criteria of chronicity and severity. This basis for subject selection was necessary because of the difficulty in obtaining a homogenous subject group on the basis of current investigative techniques, which are unable to identify a definitive cause for the pain in the majority of patients.\textsuperscript{42} For inclusion in the pain group, the subjects were required to have LBP of insidious onset of at least 18 months duration for which they had sought medical or allied health
treatment. The pain was to be of sufficient intensity to cause them to restrict their activity, involving a minimum of 3 days absent from work and be episodic with at least one painful episode per year or of a semicontinuous nature with periods of greater and minimal pain. The subjects were to have minimal or no pain at the time of testing. The mean duration of symptoms was 8.6 years (range, 2–30 years), and time off work was 9 days (range, 3–40 days). Subjects were excluded if they had neurologic symptoms, observable spinal deformity (e.g., 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 Baeeke et al.9 The demographic data and habitual activity data for both subject groups are presented in Table 1. The study was approved by the Medical Research Ethics Committee of The University of Queensland.

Experimental Design. The design of this research aimed to provide a model for the evaluation of the control of stability of the spine in response to a self-generated perturbation to the spine in control subjects and those with LBP. The model, modified from a paradigm commonly used to identify central nervous system control of body equilibrium,6,7 involved the identification of the timing of onset of electromyographic (EMG) activity of the abdominal and the lumbar multifidus muscles in association with rapid movement of an upper limb in response to a visual stimulus (Figure 1). The relationship between the action of the muscles and the direction of the reactive forces was evaluated by performance of limb movement in each of three directions. Two parameters associated with specific LBP populations, namely reduced limb movement velocity,98 and certain postural deviations,99,104 are known to influence anticipatory postural reactions.105,106 Measurement of both these factors was performed in a separate session to identify if variation existed between groups that could explain the hypothesized changes in the anticipatory trunk muscle response with LBP.

Electromyographic Recordings. A combination of fine-wire and surface EMG electrodes were used. Bipolar fine-wire electrodes were fabricated from Teflon-coated stainless steel wire (75 μm, 1 mm insulation removed; A-M Systems Inc., Everett, WA) inserted into a bevelled-edged hypodermic needle (0.70 x 32 mm) and bent back sharply against the needle, leaving the receptive ends staggered at 1.5 and 3 mm. All fine-wire electrodes were inserted under the guidance of real-time ultrasound imaging (Advanced Technology Laboratory, Bothel, WA) using a 5-MHz curved array sound head. This technique has been described in detail elsewhere.10 Electrode insertion was preceded by the application of anesthetic EMLA cream (Astra Pharmaceuticals, North Ryde, NSW, Australia). Ag/AgCl bipolar surface electrodes were used with an interelectrode distance of 12 mm. EMG data were sampled at a rate of 2000 Hz with 12-bit analog to digital conversion and bandpass filtered at 20–1000 Hz. All data were stored on computer for later analysis.

The fine-wire electrodes were inserted into the left TrA, obliquus abdominis internus (IO), and obliquus abdominis externus (EO) at standard sites: TrA 2 cm anterior to the proximal end of a line drawn vertically from the anterior superior iliac spine (ASIS) to the rib cage; IO 2 cm medial and superior to the ASIS; and EO midway between the iliac crest and rib cage in the mid-axillary line. Confirmation of placement was achieved by gentle traction of the wire and visualization of movement using the ultrasound. Surface electrodes were positioned: centrally over the muscle bellies of the anterior, middle, and posterior portions of the right deltoid muscle parallel with the muscle fibers; RA either side of a line joining the right and left ASIS, 2 cm lateral to the midline; left lumbar multifidus (MF) at the L4–L5 interspace 2 cm lateral to the spinous process. The ground electrode was placed over the right ASIS. Before attachment, the skin was prepared in a standard manner to reduce the skin impedance below 5 kΩ.

Procedure. The experimental procedure was identical for the LBP and control groups. To standardize the test position, the subject stood on a Balance Performance Monitor (SMS Healthcare, UK) (Figure 1) that provided auditory feedback of right–left weight inequality. Furthermore, the EMG activity of the muscles evaluated was monitored throughout the procedure, and the subjects were encouraged to relax consciously if the degree of muscle activity increased above a resting level. Both subject groups were able to achieve this criteria.

Table 1. Demographic Data for the Control and LBP Groups (Mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Non-LBP</th>
<th>LBP</th>
<th>F Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>28.5 ± 0.4</td>
<td>30.4 ± 7.8</td>
<td>0.08 (NS)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>173.3 ± 10.7</td>
<td>174.1 ± 8.4</td>
<td>0.05 (NS)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>67.3 ± 10.7</td>
<td>73.5 ± 11.8</td>
<td>2.97 (NS)</td>
</tr>
<tr>
<td>Activity index</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Work</td>
<td>2.4 ± 0.4</td>
<td>2.8 ± 0.8</td>
<td>2.96 (NS)</td>
</tr>
<tr>
<td>Sport</td>
<td>3.1 ± 0.5</td>
<td>3.3 ± 0.9</td>
<td>0.63 (NS)</td>
</tr>
<tr>
<td>Leisure</td>
<td>2.9 ± 0.8</td>
<td>3.0 ± 0.6</td>
<td>0.05 (NS)</td>
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</table>

LBP = low back pain; NS = not significant.
Table 2. Time Between Onset of EMG Activity of the Trunk Muscles and the Prime Mover of the Limb (Mean ± SD) for the LBP and Control Subjects*

<table>
<thead>
<tr>
<th></th>
<th>Flexion</th>
<th>Abduction</th>
<th>Extension</th>
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<tbody>
<tr>
<td></td>
<td>Non-LBP</td>
<td>LBP</td>
<td>Non-LBP</td>
</tr>
<tr>
<td>TrA</td>
<td>-38.9 ± 30</td>
<td>126 ± 104</td>
<td>-24.17 ± 25</td>
</tr>
<tr>
<td>IO</td>
<td>28.1 ± 52</td>
<td>79.8 ± 83</td>
<td>-11.24 ± 55</td>
</tr>
<tr>
<td>EO</td>
<td>58.5 ± 47</td>
<td>92.2 ± 42</td>
<td>18.7 ± 62</td>
</tr>
<tr>
<td>RA</td>
<td>84.0 ± 58</td>
<td>124.1 ± 95</td>
<td>29.9 ± 48</td>
</tr>
<tr>
<td>MF</td>
<td>9.3 ± 32</td>
<td>11.0 ± 31</td>
<td>56.8 ± 54</td>
</tr>
<tr>
<td>F value</td>
<td>13.681</td>
<td>8.811</td>
<td>5.014</td>
</tr>
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</table>

* A negative value indicates contraction prior to the prime mover.

The evaluation of timing of onset of EMG activity was conducted by computer, using an algorithm modified from that used by Di Fabio. The EMG data were full-wave rectified, filtered with a 50-Hz sixth order elliptical low-pass filter, and then analyzed using MATLAB (The MathWorks, Natick, MA) software to identify the point where the mean amplitude of 50 consecutive samples (25 msec) exceeded the mean baseline activity by two standard deviations. The mean of the baseline was calculated from the 50 msec before the warning stimulus. Each onset time was checked visually to identify trials in which the onset was disrupted by a heart beat or movement artifact. The time between the onset of contraction of both of the trunk muscles and the prime mover of the limb, i.e., the difference time, formed the basis of the analysis. Consistent with previous studies, the onset of trunk muscle activity up to 50 msec after the onset of activity of the prime mover of the limb was regarded as anticipatory. These criteria are based on the finding that the initiation of limb movement never occurs before 30 msec after the onset of activity of the prime mover of the limb because of electromechanical delay, and therefore, any activity occurring before or shortly after this point cannot be reflexly mediated.

Comparison between individual trunk muscles, between movement directions, and between subject groups involved analysis of variance (ANOVA) and Duncan’s multiple range test.

Results

Non-LBP Subject Response

When subjects without LBP flexed their shoulder, TrA was invariably the first muscle active, preceding the contraction of the anterior portion of the deltoid muscle (Figure 2 and Table 2). The reaction time for anterior deltoid with shoulder flexion was 188 ± 32 msec. Each of the trunk muscles evaluated, other than TrA, followed the onset of anterior deltoid by 9–84 msec and was significantly different from TrA. No significant difference was noted between MF and IO, IO and EO, or EO and RA. The onset of EO and RA occurred more than the 50 msec...
after the onset of the prime mover, making it impossible to rule out the possibility that the contraction of these muscles was reflexly mediated.

Abduction of the shoulder similarly resulted in contraction of TrA before the prime mover of the limb (middle deltoid; Figure 2 and Table 2). The middle deltoid was active 171 ± 54 msec after the movement stimulus. In contrast to shoulder flexion, the onset of IO also occurred before the prime mover and was not significantly different from TrA. The contraction of EO shortly after the prime mover was not significantly different from either of these muscles. Contraction of RA and MF followed the prime mover and were not significantly different from each other. The onset of MF occurred more than 50 msec after the prime mover and did not fulfill the anticipatory criteria.

Shoulder extension was associated with contraction of TrA and RA before the posterior portion of the deltoid (Figure 2 and Table 2). The latency between these muscles and the prime mover were not different to each other nor were they significantly different to the difference time of IO, which became active shortly after the prime mover. EO and MF followed the onset of activity of the prime mover and were not significantly different from each other. The reaction time of posterior deltoid was 171 ± 34 msec with shoulder extension. The onset of MF occurred outside the 50 msec anticipatory criteria.

When the each of the different movement directions was compared, no significant difference was noted between the reaction time of the muscle responsible for movement of the limb (F14.2 = 1.27; P = 0.29). However, because the sequence of activation of the trunk muscles varied between movement directions, the latency between the prime mover and each of the muscles was not consistent between directions for the majority of muscles for the control subjects (Figure 3). The difference

Figure 2. Electromyographic (EMG) data of a representative control subject for all muscles averaged over 10 repetitions for shoulder movement in different directions. The time of alignment of the traces at the onset of EMG activity of the prime mover is noted by the heavy dashed line at zero. The onset of TrA is noted by the fine dashed line. Note the onset of contraction of TrA before the other trunk muscles, and the change in sequence of RA, EO, IO, and MF as a function of limb movement direction. EMG is in arbitrary units. AD = anterior deltoid; MD = middle deltoid; PD = posterior deltoid.

Figure 3. Control group mean (filled boxes) and individual subject (joined dots) times of onset of electromyographic (EMG) activity of each of the trunk muscles relative to the onset of the prime mover of the limb with movement in different directions. Time of alignment is the onset of the prime mover at zero, denoted with the dashed line. The individual muscle is shown in the upper right corner. Note the consistent time of onset of EMG activity of TrA across movement directions and the significant differences in the other trunk muscles. F = flexion; A = abduction; E = extension. *P < 0.01.

time for IO (F14.2 = 4.62; P = 0.05), EO (F14.2 = 3.53, P < 0.05), RA (F14.2 = 16.17; P < 0.01), and MF (F14.2 = 17.25, P < 0.01) varied between directions. IO and EO were active later relative to the prime mover with shoulder flexion compared with the other movement directions; the onset of RA was significantly different between all movement directions being active earliest in shoulder extension and latest in shoulder flexion; and MF was active significantly earlier in shoulder flexion than the other two directions of movement. In contrast, no significant difference was recorded for TrA (F14.2 = 1.84; P = 0.18) across movement directions, indicating that the contraction of TrA was not influenced by the direction of the reactive forces.

**LBP Subject Response**

In contrast to the control group, when subjects with LBP performed rapid flexion of the shoulder, none of the
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Figure 4. Electromyographic (EMG) data of a representative subject with low back pain for all muscles averaged over 10 repetitions for shoulder movement in different directions. The time of alignment of the traces at the onset of EMG activity of the prime mover is noted by the heavy dashed line at 0. The onset of TrA is noted by the fine dashed line. Note the onset of TrA after the prime mover, in contrast to the control group, and the change in time of onset of TrA between directions of movement. EMG is in arbitrary units. AD = anterior deltoid; MD = middle deltoid; PD = posterior deltoid.

muscles tested was active before the prime mover, although the reaction time of the prime mover was consistent between subject groups (188 ± 53) (F14.1 = 0.0; P = 0.99). The onset of contraction of TrA, occurring after the prime mover, was not significantly different from IO, EO, or RA (Figure 4 and Table 2). MF was active significantly earlier than the other muscles and was the only muscle active within the 50 msec anticipatory criteria.

Similarly, with shoulder abduction, no muscle was active before the prime mover, and all muscles were not significantly different from each other (Figure 4 and Table 2). The prime mover reaction time was 212 ± 83 msec, consistent with the control group (F14.1 = 2.46; P = 0.13). Each of the muscles was active within the 50 msec anticipatory criteria.

Consistent with the other two movement directions for the LBP group, when shoulder extension was performed, no muscle was active before the prime mover of the limb (Figure 4 and Table 2). TrA was significantly later than IO, and MF was significantly later than RA and IO, otherwise the onset of the trunk muscles were not significantly different from each other. The prime mover was active, with a reaction time of 212 ± 57 msec, and unlike the other movements, it was delayed compared with the control group (F14.1 = 5.72; P < 0.02). The onset of TrA and MF occurred more than 50 msec after the prime mover and failed to satisfy the anticipatory criteria.

Similar to the control group, the reaction time of the prime mover was not significantly different between movement directions (F14.2 = 0.80; P = 0.46). In contrast to the control group, the difference time of TrA varied with the direction of limb movement along with the other trunk muscles (TrA: F14.2 = 8.66, P < 0.01; IO: F14.2 = 7.39, P < 0.01; EO: F14.2 = 7.76, P < 0.01; RA: F14.2 = 11.64, P < 0.01; MF: F14.2 = 14.49, P < 0.01; Figure 5). The onsets of RA, EO, IO, and MF responded in the same manner as the control group. TrA was active significantly later with shoulder flexion compared with the other movements.

Comparison of Groups
The level of background activity was low for the LBP and control groups (note background activity in Figures 2 and 4). This low activity indicates that any difference identified between the subject groups cannot be attributed to preexisting activity in the muscles or a failure to identify the onset of muscle activity resulting from a low signal-to-noise ratio.

The most consistent difference between the LBP and control groups is the significant delay in the onset of TrA in the subjects with LBP with movement in each direction.

Figure 5. Low back pain group mean (filled boxes) and individual subject data (joined dots) times of onset of electromyographic (EMG) activity of each of the trunk muscles relative to the onset of the prime mover of the limb with movement in different directions. Time of alignment is the onset of the prime mover at 0, denoted with the dashed line. The individual muscle is shown in the upper right corner. Note the significantly earlier onset of TrA with shoulder extension in contrast to the control group and the differences in time of onset of EMG of the other trunk muscles consistent with the control group. F = flexion; A = abduction; E = extension. *P < 0.01.
been addressed previously in the literature. The mechanical effect of trunk muscle contraction was not evaluated in this study. However, the results provide evidence that the central nervous system initiates contraction of the muscles of the trunk in advance of limb movement. Consistent with previous reports of anticipatory activity associated with limb movement,5,7,9 it can be implied that this activity is involved in the control of the dynamic forces associated with the movement.

Normal Neuromotor Response of the Trunk Muscles Accompanying Limb Movement

The results of the control subjects confirm the findings of our previous study of the temporal sequence of trunk muscle activity associated with movement of the upper limb (Hodges and Richardson, in preparation). The frequent contraction of the trunk muscles in anticipation of the initiation of the prime mover of the limb in the control subjects is consistent with previous studies, indicating contraction of ES and EO in advance of upper limb flexion1,5,7,61 and RA before extension.4,17 The contraction of TrA before the prime mover of the limb has not been reported previously. However, the contraction of TrA before the other abdominal muscles is consistent with the findings of Cresswell et al,11 which indicated a similar sequence of activation during ventral trunk loading. Unlike trunk loading, the activation of TrA and the other trunk muscles in anticipation of the limb movement cannot be reflexly mediated and must be preprogrammed by the central nervous system to precisely oppose the perturbing force.7 It is well accepted that preprogrammed postural muscle activity is initiated either as part of the motor command for movement of the limb7 or parallel with the motor command.25

The variation of the reactive forces associated with different directions of limb movement can be anticipated and accounted for by the preprogrammed response,7,16 providing an explanation for the variation in sequence of muscle contraction reported between movement directions. The contraction of MF earlier with upper limb flexion than extension is consistent with this proposal to maintain the alignment of the trunk against the reactive force producing flexion of the spine. Correspondingly, the reversed direction of the reactive forces with shoulder extension is consistent with the contraction of the trunk flexors (RA, EO, and IO) earlier with upper limb extension than the other movement directions to control the resultant trunk extension moment. The consistently early activation of EO, IO, RA, and MF with limb abduction is consistent with the requirement to control the lateral flexion moment of the trunk expected to accompany movement in this direction. This variation in time of onset of each of the trunk muscles with variation in the movement direction indicates that the response is not simply a general increase in background activity but is related specifically to the anticipated perturbation.

Comparison of Postural Parameters and Limb Movement Velocity

No difference in mean or peak arm velocity or either of the postural parameters was noted between the two groups (Table 3), which indicates that the difference noted between the two subject groups cannot be explained by variation in these parameters.

Discussion

The model has identified a consistent dysfunction of the motor control of TrA in people with LBP that has not

Table 3. Postural Parameters and Lumbar Flexion Velocity for Control and LBP Subjects (Mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Non-LBP</th>
<th>LBP</th>
<th>P Value</th>
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<tbody>
<tr>
<td>Lumbar lordosis (*)</td>
<td>23.1 ± 7.0</td>
<td>28.8 ± 5.5</td>
<td>1.03 (NS)</td>
</tr>
<tr>
<td>Pelvic tilt (*)</td>
<td>12.4 ± 4.4</td>
<td>14.5 ± 5.4</td>
<td>1.32 (NS)</td>
</tr>
<tr>
<td>Peak velocity (°/sec)</td>
<td>515.7 ± 68.0</td>
<td>469.1 ± 97.0</td>
<td>1.51 (NS)</td>
</tr>
<tr>
<td>Mean velocity (°/sec)</td>
<td>309.1 ± 65.5</td>
<td>235.7 ± 57.9</td>
<td>5.83 (NS)</td>
</tr>
</tbody>
</table>

LBP = low back pain; NS = not significant.
In contrast, the time of onset of contraction of TrA in the control group was not affected by the direction of limb movement and the associated reactive forces, which suggests that contraction of this muscle is related to either the control of a parameter that remains consistent across movement directions or acts via a mechanism that permits only nondirection-specific control. Although TrA cannot contribute to the production of sagittal plane motion, it has been suggested to be involved in production of trunk rotation. The time of onset of TrA did not vary between limb flexion and extension, which would provoke rotation in opposite directions, which suggests that it is unlikely that this is the parameter being controlled by this muscle, although evaluation of the magnitude of the EMG response would clarify this issue. Consequently, the contraction of TrA is hypothesized to contribute to the control of the forces associated with limb movement by increasing the stiffness of the lumbar spine through raising the intra-abdominal pressure and tensioning the thoracolumbar fascia, which has been described previously, rather than the control of direction-specific aspects of the reactive forces. The results of the study are consistent with this hypothesis. The proposed increase in stiffness of the lumbar spine in anticipation of limb movement would limit intersegmental translation and rotational forces produced by the perturbation to the spine and may provide a more stable lever over which the other trunk muscles can act. This is essential because buckling of the spine has been shown to occur when loaded by as little as 20 N. The mechanical output of the anticipatory contraction of TrA is to be evaluated to confirm this hypothesis and is the focus of ongoing research.

**Dysfunction of Motor Control of the Trunk Muscles in Patients With Low Back Pain**

The delay in onset of contraction of trunk muscles associated with movement of the upper limb in patients with LBP indicates a significant deficit in the automatic motor command for control of disturbance to the spine. Although each of the trunk muscles evaluated demonstrated some difference in onset time between groups, TrA demonstrated the most significant deviation. The unexpected finding that TrA adopted a direction-specific response indicates that the change is not simply a delay but a fundamental problem of motor control of this muscle. Additional research is required to determine if the dysfunction precedes or follows the onset of LBP.

Considering the hypothesized role of TrA in the generation of spinal stability, the consequence of the delay in contraction of this muscle would be reduced spinal stiffness at the time of initiation of the limb movement and the resulting reactive forces. Irrespective of the maximal strength or endurance capacity of this muscle, the contraction would be ineffective in optimally controlling these forces. Because of the inherent instability of the lumbar spine, failure of muscular stabilization produces an increased risk of injury to the spine. Only 2° of intersegmental rotation is required to produce micro-trauma of the structures of the lumbar spine. The potential for this to occur as a result of delayed stiffening of the spine is significant. The delay in onset of contraction of each of the other muscles in only one movement direction indicates a failure of these muscles to fulfill their trunk-supporting role in association with reactive forces in certain directions only. The consequence of the slightly earlier activation of MF with shoulder abduction in the LBP group is uncertain.

**Potential Mechanisms for Temporal Delay in the Trunk Muscles**

The only other studies that successfully have identified a similar delay in the onset of postural muscles in association with rapid limb movement have been the examinations of people with central nervous system disorders such as a lesions of the frontal lobe and Parkinson's disease. The potential for dysfunction of the central nervous system to explain the mechanism for the delay in TrA and the other abdominal muscles cannot be excluded with the current methodology. Other studies have identified a relationship between LBP and changes in central nervous system parameters, including a loss of the biphasic pattern of contraction of MF when catching a load anteriorly, increased postural deviation, reduced reaction time in a finger movement task, and a high incidence of neurologic signs, such as changes in muscle tone and coordination.

Other factors also may produce deviations in the timing of onset of muscle contractions, such as the previously mentioned delay in postural muscle onset resulting from decreased limb movement velocity. Because the limb movement velocity was comparable between groups, this is unlikely to explain the present findings. Other parameters such as reflex inhibition caused by effusion, pain, ligament stretch, or capsular compression may influence the timing of activation of the trunk muscles by lowering the excitability of the motoneuron pool. This would result in an increased time taken to exceed the threshold, producing a delay in contraction onset. Fatigue and postural variation also have been suggested to influence the excitability of the motoneuron pool. However, it is uncertain if the recorded 61-165 msec delay in onset of contraction of TrA or the changes in the directional specificity of the response could be explained by this mechanism.

The potential for other central mechanisms to explain the delay in EMG onset of TrA cannot be excluded with the current data and other explanations may exist, including the potential for the delay to be compensatory to the pain. Additional studies are required to evaluate this phenomenon in more detail to elucidate the mechanism and consequence of the delayed EMG onset.
Methodologic Considerations

No analysis of segmental or general spinal motion was involved in the current study, potentially limiting the extrapolation of findings to the lumbar stabilization mechanism and the consequence of the delayed timing of the trunk muscles. However, only small deviations are required to produce significant microtrauma to the spine, and current methods of movement analysis are unable to provide appropriate discrimination to evaluate this factor.

To evaluate an isolated component of motor control, it was necessary to involve a movement that was not functional. Slight differences in temporal parameters have been reported in this type of movement compared with natural movements, and extrapolation of findings to function must be done with caution. However, it must be acknowledged that the deficit of TrA identified using this model indicates a fundamental problem of motor control of this muscle and is likely to be reflected in alternative movement types. This is an area of ongoing research.

The potential for cross-talk between the abdominal muscles must be considered because the width of TrA, IO, and EO range between 0.4–1.5 cm. The significant difference in the timing of onset of EMG activity of each of the abdominal muscles in the majority of patients indicates that cross-talk was minimally significant in the current study.

Finally, the evaluation of onset of EMG activity using fine-wire electrodes is likely to have produced a slight inaccuracy in onset determination. Because the innervation zone of TrA, EO, and IO has not been identified, the distance between this point and the receptive end of the electrode is unknown and likely to have been variable. The result of the variable distance and the slow conduction velocity of the action potential along the muscle fiber (3–5 m/sec) is a delay in the detection of onset by a variable period of up to 30 msec if the distance is 0.1 m. The detection of EMG onset is not influenced to the same extent when recordings are made using surface electrodes with a larger receptive area. However, the result of this mismatch in recording accuracy would be expected to produce similar variation for both subject groups and cannot explain the delay of 61–165 msec.

Conclusion

The function of TrA has been largely ignored in the evaluation of spinal stabilization and protection. Furthermore, the role of this muscle in lumbar spine dysfunction has not been addressed. The results of this study provide several potential implications for basic science and clinical research:

1. The proposed significance of TrA to the lumbar stability mechanism suggests that future modeling of the spine should include investigation of this muscle;
2. The model may provide a method to evaluate objectively dysfunction of the motor system and changes in this system over time with disease progression or rehabilitation;
3. The possible identification of a motor control deficit provides the impetus for additional evaluation of the mechanism and other possible manifestations of dysfunction of this system; and
4. The observed deficit may provide a basis for the development and evaluation of rehabilitative and preventative strategies for patients with LBP, focusing on the resolution of the anomaly and restoration of normal function. Additional clinical investigation is required to evaluate this hypothesis.

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